THE SURGICAL CLINICS OF NORTH AMERICA

DECEMBER 1977

COMPLICATIONS OF GENERAL SURGERY

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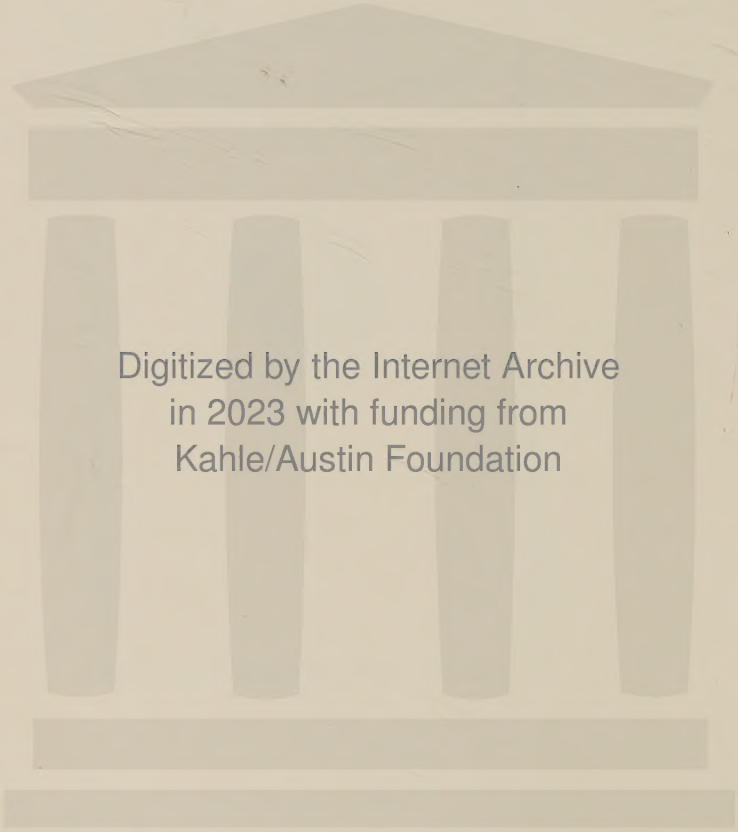
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THE SURGICAL CLINICS OF NORTH AMERICA

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SYMPOSIUM ON
COMPLICATIONS OF GENERAL SURGERY

Robert J. Freeark, M.D., *Guest Editor*

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Foreword

"If it wasn't for bleeding, everybody would do surgery!" This quotation, stolen from a friend and colleague, is both a humorous and at times consoling reminder of the part that "complications" play in our surgical lives. While many complications arise during or shortly after the operative procedure, the ground work for many "adverse events" is often laid by omissions and errors prior to the operation.

In this issue of the *Surgical Clinics of North America*, surgeons from the Greater Chicago Area have tried to share with their colleagues some of their experiences with the varied "problems" of our craft. Each of these authors is a respected clinical surgeon, active in residency training, and accustomed to the frank and open discussion of complications during the weekly Morbidity and Mortality Conference that is the hallmark of the true teaching hospital.

In some papers the subject matter is based largely on a review of an institution's experience with a particular operation or disease. In other articles, the authors have clearly "searched" the available medical literature to attempt to identify causes, pitfalls, and alternative solutions to the complications of surgery. In each article, the surgeon has been urged to be concise and practical, and yet provide the little details that are so often the key ingredient to a successful result. To this task they have responded admirably, and each of them shares with me the sincere hope that the information will serve you and your patients well.

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Monitoring in Critically Ill Patients

*Robert J. Baker, M.D.**

Most critically ill patients are salvageable if cardiac output, pulmonary perfusion and diffusion, and renal parenchymal blood flow can be maintained at reasonable levels. Evaluation of the critically ill should be carried out promptly and with organization (Table 1).

URINARY TRACT CATHETERIZATION

Placing a catheter in the bladder, with measurement of minute or hourly urine volume, is a helpful method for monitoring renal blood flow, if the kidney can concentrate. Generally speaking, catheterization of the urinary bladder in the female has few if any significant complications, although the use of a Foley catheter, especially in diabetic patients, may result in significant trigonitis, cystitis, and even ascending urinary tract infection. In the male, every attempt should be made to use the smallest possible urethral catheter, 12 French size being perfectly satisfactory, and such a catheter should not ordinarily be used with the Foley balloon inflated. In the event that the patient is uncooperative or irrational and manages to remove the catheter, it is a simple matter to place a moderate size stitch in the inner aspect of the thigh, and tie the catheter end protruding from the urethra to the skin of the thigh, thereby discouraging such removal. If the patient is not agitated, or is comatose, the suture is not necessary; an effective device for holding the straight urethral catheter in place can be fashioned from water-resistant adhesive tape and several stutures tied around the tip of the catheter, fastening these to the tape plastered to the inner aspect of the upper thigh. If the catheter is to be left in place for only a short period of time, and the patient is conscious and alert, the use of a 10 ml Foley balloon has little associated difficulty.

Of greater import, however, is the patient with lower abdominal trauma of pelvic fracture, in whom a real possibility of trauma to the urethra exists.[3] Approximately 8 to 10 per cent of these patients will

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Table 1. *Modalities to be Monitored in Critically Ill Patients*

1. Urine specimen: urethral catheter or cystostomy
   a. Urine: volume, osmolality (specific gravity)
2. Infusion pyelogram
3. Central venous pressure
4. Pulmonary artery end diastolic pressure, pulmonary capillary wedge pressure
5. Cardiac output
6. Arterial pH, Po_2 and Pco_2
7. Intra-arterial pressure
8. Serum osmolality
9. Abdominal paracentesis
10. Visceral radionuclide scanning
11. Angiography (arteriography, venography)
12. Blood determinations
    a. Glucose
    b. BUN, creatinine
    c. Hematocrit, platelet count
    d. Fibrinogen, prothrombin time
    e. Partial thromboplastin time
    f. Electrolytes

have sustained significant urethral trauma with major pelvic fracture, and urethral interruption may have occurred. The signs of severe urethral injury are: (1) the prostate is not palpable on digital rectal examination (the prostate floats up and out of the pelvis with posterior urethral transection); (2) in attempting to void, frank blood is passed, or a few drops of blood are seen at the urethral meatus; (3) the conscious patient attempts to void, and, although the bladder is palpable, is unable to do so; (4) the patient passes a small or modest amount of bloody urine, which clears terminally in the process of urination. If catheterization is attempted with such a ruptured urethra, several significant anatomic problems may be precipitated: (a) the tear in the urethra may be enlarged, leading to a more extensive laceration than was present initially; (b) a false passage may be made, leading to contamination of the periurethral tissues and the perivesical space with bacteria which will cause extensive inflammation and fibrosis; (c) some bloody urine may be obtained, leading to a delay in suspecting and establishing the actual anatomic diagnosis. Any of these changes may herald severe stricture, but the presence of periurethral infection with the extensive scarring which results is certain to make stricture an anatomic reality; the repair of such an infected post-traumatic stricture is extremely difficult, if not impossible, and the alteration in function of the lower urinary and genital tracts is catastrophic.

Patients who have sustained lower abdominal or pelvic injury should always be asked to void prior to the insertion of a urethral catheter, plain or with a Foley balloon. If the patient cannot void, or voids a small amount of blood or bloody urine which clears, or in an unconscious patient with such injuries who does not void on request, a *retrograde urethrogram* should be performed prior to insertion of a catheter into the urethra and bladder. This may be done with a small balloon-tipped urethral catheter in the terminal urethra, where the

balloon is inflated and is then grasped with the thumb and index finger of the operator. Alternatively, a sterile, smooth-tipped, bulb syringe may be inserted directly into the urethra, and the urethra secured around the syringe by finger pressure. Twenty-five ml of sterile water-soluble dye, such as 30 per cent diatrizoate, is then injected slowly into the urethra. Oblique and lateral x-rays of the urethra and lower pelvis will demonstrate tears in the urethra (portable films may be used). With urethral injury, suprapubic cystostomy should be undertaken immediately to empty the bladder and monitor urine output thereafter. If the urethra is seen to be intact, a catheter may be inserted into the bladder and cystography undertaken in order to rule out bladder disruption as the source of blood. The urethrogram should always be performed prior to cystogram, since filling the bladder with dye will result in confusion as to the site of the tear if there is any extravasation of dye. Bladder injury can easily be confused with high posterior urethral rupture, since dye from the proximal urethra frequently enters the perivesical space. Although it may seem unnecessary to proceed to cystostomy in a patient with a relatively small urethral rupture, this course is safer than attempting manipulation of the damaged urinary tract with a urethral catheter.

INFUSION PYELOGRAPHY

Roentgenographic visualization of the upper urinary tract has become a commonly used study in patients admitted with abdominal or thoracic trauma, either blunt or penetrating. It has been usual practice to administer 100 to 150 ml of 50 per cent diatrizoate in the first liter of intravenous fluid infused, following which the first abdominal film taken when the patient is resuscitated serves as an intravenous pyelogram. There is a need under these circumstances to ensure that both kidneys are present, that the renal parenchyma is intact, and that there is no delayed or incomplete visualization on either side.

Indiscriminate administration of these doses of dye in patients in shock, or profoundly fluid depleted, may lead to serious impairment of renal function. It is not unusual to find patients with creatinine levels above 2 mg per dl following this study, and, although the creatinine returns to a normal level quite rapidly following successful resuscitation, there is a distinct possibility that chronic renal disease may be severely and adversely affected by the indiscriminate use of dye. For that reason, it is now advocated that the administration of intravenous dye for purposes of visualizing the urinary tract be delayed until resuscitation is well underway, the patient is known to be well hydrated, and urine output has increased. In addition, the use of mannitol, without loop diuretics (furosemide), will hasten the excretion of dye and lessen the exposure time of the renal parenchyma to this toxic substance. No pyelograms should be done without obtaining an adequate history of drug sensitivity, specifically to iodide-containing dyes, either from the patient or a member of his family. Significant cardiovascular or pulmonary complications may ensue following administration of even relatively small amounts of dye to which the patient is allergic.

CENTRAL VENOUS PRESSURE (CVP)

CVP monitoring has been widely employed, with major emphasis on its value in prevention of circulatory overload with colloid or crystalloid. In addition, the introduction of medications, a route for hyperalimentation into the central venous system, and the possibility of inserting a venous pacing electrode through the CVP catheter have further popularized its use. Unfortunately, the interpretation of the measured central venous pressure has been shown in more recent years to be erratic at best, and frequently grossly misleading. An elevated central venous pressure may be found in hypovolemia, while the patient is still fluid depleted, if the patient is on positive pressure ventilation, if there is cardiac tamponade, with general anesthesia (if cardiodepressant anesthetic agents are used), or with preexisting or traumatic cardiopulmonary disease. Equally misleading is the well-recognized fact that patients may be grossly overloaded with fluid, especially crystalloid, to the point of pulmonary edema and a normal central venous pressure reading may still be obtained. An isolated reading is meaningless, but the CVP response to intravenous fluid therapy is the primary concern.[8, 22]

Complications

Complications of central venous pressure monitoring are mechanical, due either to the placement technique or to the method of management of the catheter once it is in place (Table 2). *Phlebitis* at the site of insertion of the venous catheter is much commoner when the catheter is inserted into the median basilic, cephalic, or brachial veins in the arm than when the catheter is placed via the subclavian or jugular veins percutaneously. Obviously, no catheters should be inserted under any except the strictest possible aseptic conditions. The site for cutdown or

Table 2. *Complications of Venous Indwelling Catheter Placement*

COMPLICATION	INCIDENCE
1. Phlebitis at insertion site	30–40 per cent
2. Venous thrombosis	10–20 per cent
3. Catheter sepsis	5–15 per cent
4. Pneumothorax	2–10 per cent
5. Vein injury	2– 5 per cent
6. Hemothorax	2– 5 per cent
7. Chylothorax	< 1 per cent
8. Pulmonary embolism	1– 4 per cent
9. Catheter embolism	Rare
10. Perforation of the atrium, ventricle	Rare
11. Mediastinal effusion	Rare
12. Hydrothorax	< 1 per cent
13. Cardiac tamponade	Rare
14. Air embolism	< 1 per cent
15. Brachial plexus, arterial injury	Rare
16. Hyperosmolar coma	Rare
17. Unilateral pulmonary edema	Rare

percutaneous insertion should be carefully prepared for 10 minutes with an organic iodine skin antiseptic; meticulous surgical technique is necessary to ensure that catheter placement is performed with the same care as any other operative procedure. This requires the use of mask, sterile gloves, sterile gown, adequate drapes, light, and instruments. The catheter site is ordinarily redressed once daily, at which time all dressings are removed to the point of insertion of the catheter, the skin is cleaned with a defatting agent, and fresh 0.1 per cent gentamycin ointment is applied to the catheter insertion site. In the event that a cutdown has been required in the periphery, the catheter should not be brought out through the primary incision, but through a tiny stab wound distal to the incision, so that the catheter traverses a subcutaneous tunnel prior to its entry into the vein. Furthermore, the catheter should be carefully fixed to the skin by two loosely-looped anchor sutures at the site of the stab wound, and there is no possibility of the catheter inadvertently advancing beyond the point of initial insertion.

Venous thrombosis is most likely to occur if the catheter size approximates that of the vein in which the catheter resides, if there is excessive motion of the catheter in a peripheral vein, or if irritating substances in high concentration are injected through the venous catheter. A firmly anchored, small catheter which is dressed with adequate care on a daily basis can often be used for monitoring throughout the acute course. The incidence of venous thrombosis is primarily related to the time in which the catheter is indwelling, any period exceeding 72 hours being associated with a higher incidence of thrombosis than are shorter periods.

Catheter sepsis is a frequent occurrence of CVP catheterization, primarily in those patients in whom intravenous hyperalimentation is administered through the catheter. If blood is withdrawn through the central venous pressure catheter, there is likewise a considerable increase in the frequency with which catheter sepsis occurs.[20] It is not necessary to use amphotericin or other antibiotics specifically for the purpose of protecting the patient against sepsis, but any sign of unexplained systemic infection requires immediate removal of the catheter, and culture of the tip of the catheter (both aerobic and anaerobic media should be inoculated). With subsidence of evidence of infection, it is reasonable to reinsert a CVP catheter at another site some 24 hours later. Appropriate antibiotics should be administered for 7 days for any organisms which are shown bacteriologically to be implicated.

Pneumothorax occurs primarily with subclavicular puncture, although it has been observed on rare occasion with internal jugular cannulation.[21] Since the apex of the pleura lies directly behind the subclavian vessels, pneumothorax of greater or lesser proportion can result from perforation of the apical pleura and/or lung (Fig. 1). A routine postinsertion x-ray, which is always taken, revealed the 25 per cent pneumothorax demonstrated. Simple needle aspiration of the pleural space may suffice; if not, a thoracostomy tube is required. As long as adequate fluctuations of the saline column are noted with respiration, it is not necessary to remove the catheter. If these are absent, or fluid cannot

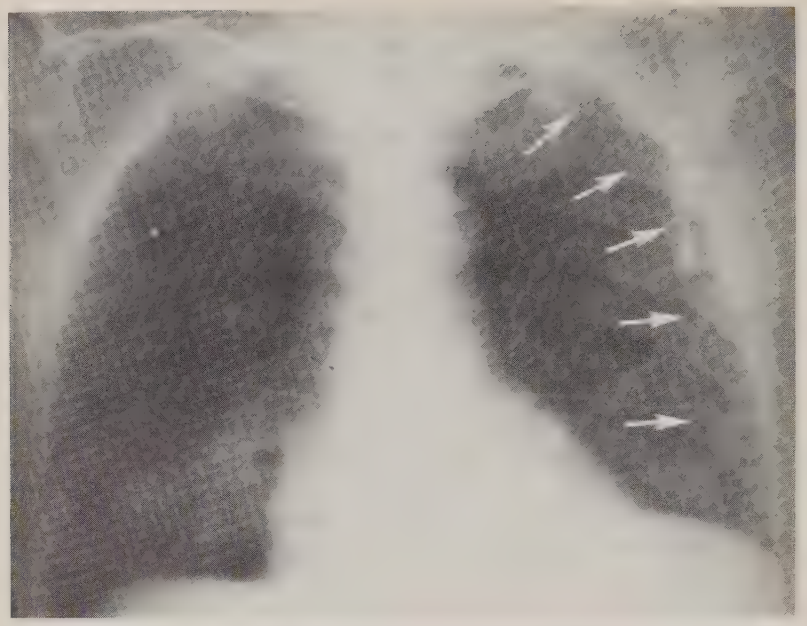

Figure 1. This PA roentgenogram of the chest was obtained 60 minutes after the insertion of a central venous pressure catheter percutaneously through the left subclavian vein. The needle has been withdrawn from the skin and held to the catheter by a plastic collar. The CVP catheter is seen to progress toward the heart at the left aspect of the superior mediastinum, but 5 ml of 50 per cent diatrizoate was injected through the catheter when the next film was obtained to localize the tip of the catheter. There is a 25 per cent pneumothorax (arrows demonstrate the lateral border of the lung), which was promptly aspirated and did not recur.

be freely infused, the catheter should be removed and reinserted at another site.

Vein injury and *hemothorax* are a consequence of the insertion of the needle through which the subclavian or internal jugular catheter is to be placed beyond the posterior wall of the vein into the soft tissue or pleural space. Hemothorax may occur from a tear of lung parenchyma at the apex and is analogous to the pneumothorax previously described. It is surprising that these two complications are not more frequent, since a significant percentage of attempted percutaneous cannulations does result in difficulty, such as multiple venipunctures to assure free flow of venous blood, or even inability to properly cannulate the vein, resulting in abandonment of the procedure. The latter course is preferable to injudicious repeated attempts to insert the cannula, and common sense would dictate that two unsuccessful attempts to cannulate either of these veins should cause termination of the procedure at that site and resort to another route of access to the central venous circulation. Pressure will control venous bleeding under almost all circumstances, although it is not uncommon for such trauma to result in venous thrombosis in the subclavian vein. In the event of hemothorax, the insertion of a chest tube may be necessary to free the pleural space of the blood as well as to indicate if and when bleeding has recurred.

Chylothorax is rarely seen, but may occur in cannulation of the left jugular vein or, occasionally, with the supraclavicular approach to the left subclavian vein. Rarely, chylothorax could occur if the catheter traversed the superior cava, then exited into the mediastinum and lacerated the thoracic duct. A thoracostomy tube allows identification of chyle, and if the chyle leak persists, operative ligation of the thoracic duct in the chest is required.

Pulmonary embolism was somewhat more common when polyethylene catheters were the type used most often for central venous pressure monitoring. Since other softer types of catheters have been adopted for routine use (polytetrafluoroethylene, etc.), the incidence of major thrombus formation at the tip of the catheter is much less frequent. In addition, the shorter period of time in which the catheter is allowed to remain in place, the prohibition of blood sampling through the central venous pressure catheter, and the abandoning of the infusion of venous irritants has diminished the incidence of thromboembolism. It is also usual now for prophylactic small-dose heparin ("mini-heparin") administration to be used in critically ill and traumatized patients, presumably lessening the incidence of catheter thrombus formation.

Catheter embolism is a significant complication which may result in a genuine catastrophe. The reason that indwelling catheters break is threefold: first, the catheter may have an intrinsic defect, and after a greater or lesser time in situ, a piece may break off without apparent cause (probably very rare); second, the catheter may be inserted in the periphery, and the limb in which the catheter resides may be allowed free movement, such that continuous flexion and extension at the elbow, or even at the shoulder, may cause the catheter to break due to repeated stress; third and commonest, this unfortunate accident can occur when an inexperienced operator, having inserted the catheter through the needle, decides to pull the catheter back without first removing the needle from the vein. The cutting edge of the needle will shear the catheter off, following which the catheter may embolize into the central circulation, perhaps into the right heart. If the catheter breaks or is cut in the extremity, a tourniquet should be applied as high as possible in the limb, and the vein should be explored locally; it is usually possible to retrieve the catheter if this accident is discovered early.[9] In the event that the catheter has migrated into the central circulation or into the right atrium or ventricle, there are several devices for snaring these via the internal jugular or femoral vein. A wire snare has been successfully employed, and a recent report has appeared in which a catheter was removed from the right atrium with a Caves-Shulz cardiac bioptome, introduced through the femoral vein.[6] In the event that it is not possible to remove the catheter by nonoperative means, then cardiotomy is the only recourse, since allowing the catheter fragment to remain in the central venous circulation or in the heart will be likely to lead to sepsis, endocarditis, thrombosis, or perforation of the heart, with subsequent tamponade.[9]

Perforation of the atrium or ventricle has rarely been described, but may occur more frequently than has been appreciated. This is a consequence of one of two errors in technique, the usual one being fail-

ure to adequately secure the catheter at the site of insertion with a pair of sutures through the skin, which then tightly invest the catheter. If this is not done, the catheter tends with motion of the shoulder or arm to migrate in a proximal direction, and, in the case of those catheters inserted through the brachial system, even extending the arm over the head will move the tip of the catheter from 2 to 4 cm further into the central circulation. If the catheter perforates the atrium or ventricle, *cardiac tamponade* will ensue, especially since fluid will usually be infused through that catheter, commonly with a pressure infuser.[1, 2, 15, 18] The second error, less commonly made, is to introduce too long a segment of catheter, which enters the atrium or ventricle, sometimes curls up, and by necrosis perforates the myocardium. It is standard practice to confirm the position of the catheter tip as soon as it is inserted with a chest x-ray, and it is equally good practice to inject 5 ml of dye through the catheter in order to be certain that the dye does not sequester in a closed space, but is promptly removed in the rapidly moving column of blood in the vena cava. Further, the catheter tip position should be rechecked daily with portable chest x-ray, since subsequent movement of the catheter may occur, causing pressure necrosis of the atrial wall.[1, 2, 15, 29] Whenever there is sudden, unexplained deterioration in the cardiovascular system in a patient with a central venous pressure catheter in place, this suggests that one of several complications may have occurred, including perforation of some portion of the right side of the heart or of the superior vena cava.[2] Aspiration of the pericardium, accompanied by removal of the catheter entirely, usually results in satisfactory resolution of the problem, although diagnosis can be enhanced by the injection of dye through the catheter, as noted above, when a chest film is taken for suspect tamponade.[10, 35]

Approximately 64 per cent of superior vena cava catheters or innominate vein catheters are in acceptable position, but 36 per cent are unacceptably placed; these are primarily in the atrium, although an equal number are seen to enter the internal jugular vein, having turned upward into the internal jugular system, instead of progressing more centrally toward the heart.[21] Mediastinal effusion may occur in the rare instance in which the catheter has perforated the wall of the superior vena cava. If such a perforation occurs, infusion of fluid will raise mediastinal pressure to the point where it exceeds right atrial pressure, and there will be severe and prompt obstruction of flow through the great vessels and into the atrium.[10] In the event that a widened mediastinum is seen on x-ray, which is the cardinal finding in great vein injury, a parasternal pericardiocentesis should be done, in order to attempt to aspirate fluid from the pericardium and to diminish the pressure effect on right atrial filling.

Air embolism has been a rare, but not unexpected, complication of central venous pressure monitoring or infusion, when the catheter or adapter is inadvertently separated from the intravenous line. Negative intrathoracic and intramediastinal pressure with inspiration sucks air into the venous system, especially if the patient is sitting or standing. The exact volume of air required to cause symptoms, and ultimately death, is at least 40 to 60 ml, and may exceed 100 ml in some adults.

Prevention is based on meticulous attention to small detail; if the same individual(s) change dressings on all central venous pressure catheters in various patients in a hospital or unit, the actions necessary to prevent such complications as air embolism become automatic. This has stimulated the development of numerous "hyperalimentation teams," whose prime purpose has been to assume full responsibility for catheter care, including dressing change, securing of catheter-tube interfaces to prevent accidental disconnection, daily wound cultures, weighing the patient, and checking serum and urine electrolytes. Most institutions rely on a tight-fitting friction connection between the catheter adapter and the tubing, but this should be reinforced with several layers of waterproof tape, and the tubing should be adequately secured to the chest wall or to the extremity with further adhesive binding. Major troubles can develop from careless or inexperienced personnel changing the tubing and fluids daily, and this should not be permitted to occur.

Brachial plexus injury is, like arterial injury, the result of overzealous introduction of a subclavian needle in the hands of the inexperienced. In the event that cords or divisions of the brachial plexus are impaled by the needle, the occurrence of pain in the hand, arm, or shoulder should immediately alert one that the tip of the needle has progressed well beyond the vein wall. Arteriovenous damage, including *arteriovenous fistula*, is a rare but more difficult complication to detect, unless a well formed arteriovenous fistula persists. With swelling in the supraclavicular or subclavicular area, or the occurrence of distended veins in the extremity distal to the insertion of the catheter, auscultation of the supraclavicular fossa may detect the continuous machinery murmur of the arteriovenous fistula; any significant difference in venous pressure in the veins of the affected limb as compared with the opposite should make one strongly suspicious that a communication has been established between the arterial and venous sides of the circulation. An arteriogram will delineate the location of the fistula. Any fistula is a source of future difficulty, and should be dealt with surgically at the earliest opportunity. Injury to the brachial plexus, provided that a large hematoma is not present, can be treated expectantly for several days; if, however, there is a detectable hematoma in the vicinity of the brachial plexus and symptoms of nerve irritation or deficit persist, it is imperative that the hematoma be evacuated as early as possible, since permanent pressure damage of the neural structures may result.

Unilateral pulmonary edema and *cerebral hyperosmolar insult* have been reported from central venous pressure catheter infusions in which the tip of the catheter is introduced, in the first instance, into one pulmonary artery, and, in the second instance, into the internal jugular system upward toward the brain. With rapid fluid infusion into one pulmonary artery, it is possible for unilateral pulmonary edema to occur.[32] The infusion of hyperosmolal fluid into the jugular vein may precipitate hyperosmolar coma and brain damage, so that every catheter should be checked carefully prior to the infusion of any significant volume of fluid by routine x-ray studies. Obviously, such catheters must be radiopaque, or a slow infusion of dye through the catheter will allow visualization of the tip of the catheter in the rapidly flowing venous stream by fluoroscopy or x-ray.

PULMONARY ARTERY END DIASTOLIC PRESSURE, PULMONARY CAPILLARY WEDGE PRESSURE

Until the late 1960's, CVP monitoring was extensively used to monitor fluid volume, and to prevent fluid overload. It became apparent, however, that an elevated central venous pressure often reflected a failing right ventricle, rather than fluid overload, or could result from pericardial tamponade. Even more disconcerting was the fact that patients with gross fluid overload, even with pulmonary edema, often had a normal central venous pressure.

In 1970, Swan and Ganz described the use of a balloon-tipped, flow directed catheter for hemodynamic monitoring; it is possible to measure either pulmonary artery end diastolic pressure or pulmonary artery wedge pressure.[34] This device provides an accurate measure of left atrial pressure, in contrast to the CVP which reflects right atrial pressure. Left atrial pressure monitoring, with the Swan-Ganz catheter, has proven to be far more sensitive to fluid depletion or fluid overload than is the central venous pressure. Furthermore, in patients with myocardial failure, with pulmonary vascular changes, or when septic myocardial depression has occurred, the central venous pressure defies interpretation, whereas the balloon flotation catheter (Swan-Ganz) monitor of pulmonary capillary wedge pressure is an excellent device for recording pressure in the left atrium, hence left ventricular diastolic filling pressure.

There are, however, several problems with the use of the Swan-Ganz catheter which make interpretation of results somewhat difficult. When patients are on a ventilator, and positive end expiratory pressure (PEEP) is used at 10 cm of water or more, the measurement of pulmonary capillary wedge pressure (PCWP) is materially affected; when the level of PEEP reaches 15 cm of water, the catheter tip is then actually measuring airway pressure rather than left atrial pressure, since the capillary bed is compressed by the distended alveoli under those expiratory pressures.[23, 38] It is satisfactory to use pulmonary artery end diastolic pressure (PAEDP) rather than pulmonary capillary wedge pressure, unless the patient has respiratory insufficiency (ARDS) or pulmonary hypertension, both of which elevate PAEDP. If possible, PEEP should be removed from the system for 60 to 120 seconds prior to the measurement of either end diastolic pressure or wedge pressure in order to lessen the dampening effect of the increased intra-alveolar pressure.

The newer balloon flotation catheters are four lumen catheters, the third and fourth channels carrying the thermistor probe and for right atrial iced saline injection in order to determine thermodilution cardiac outputs with the same intravenous catheter that allows measurement of the capillary wedge pressure. This technique involves the injection of an aliquot of iced saline at approximately 0° C, but continuous positive pressure ventilation or intermittent mechanical ventilation, as well as positive end expiratory pressure greater than 10 cm of water, create aberrations and fluctuations in the pulmonary artery temperature pattern not observed in the patient who is not on a ventilator. This is a significant error when measuring cardiac output by the thermodilution technique; deviations of cardiac output as great as 14 per cent have

been reported if injections of the cold indicator were not very carefully timed.[38] The best way to measure cardiac output is during apnea, or, failing the ability to make the patient apneic for some seconds, the iced saline should be injected at the same point in the respiratory cycle, generally at the terminus of expiration.

If mixed venous blood sampling is to be done through the Swan-Ganz catheter (used to determine cardiac output by the Fick principle or to measure oxygen consumption), the blood must not be taken from the "wedge" position, since blood in the capillary bed is contaminated with arterialized blood; this can be checked by measuring the mixed venous concentration of CO_2, which may be equal to or lower than the arterial Pco_2 suggesting contamination of the pulmonary arterial blood by pulmonary capillary blood.[33]

Hazards and complications of the Swan-Ganz balloon flotation catheter placement are identical to those of the central venous pressure catheter with several other difficulties intrinsic in the passage of and placement of the balloon tip (Table 3). *Pulmonary infarction* follows inadvertent wedging of the catheter and balloon by uncontrolled advancing of the inflated balloon into the pulmonary artery branch, usually at a time when active monitoring is not taking place. This can occur if the brachial vein or other vessel in the upper extremity is used and the patient is allowed to move the arm, rather than to have the arm secured at the side in a stable position. The catheter tip may wander into a small branch of the pulmonary artery, even if the balloon is deflated, causing obstruction of that branch of the pulmonary artery and infarction of the distal lung.[13] When recognized, of course, the catheter should be withdrawn until the pressure wave indicates that the catheter is in the right ventricle, and then the balloon reinflated and the catheter allowed to drift into the pulmonary artery. Careful fixation of the catheter to the skin by adhesive tape, immobilization of the limb, and adequate dressing is important to prevent this particular complication.

Protracted inflation of the balloon in the wedge position in order to obtain a good tracing of the wedge pressure, may cause similar difficulty. As a rule, the balloon should not remain inflated for more than 10 to 20 seconds, at which time the balloon should be deflated and the catheter withdrawn slightly in order to be certain that flow to that segment of lung tissue is adequate. A pulmonary artery pressure tracing should be apparent. In the event that an unsatisfactory wedge pressure tracing is obtained, it sometimes helps to turn the patient from side to

Table 3. *Complications of Capillary "Wedging"*

1. Pulmonary infarction
2. Rupture of a branch of the pulmonary artery
3. Atrial, ventricular arrhythmias
4. Balloon rupture
5. Intracardiac catheter knotting
6. Superior vena cava, intracardiac thrombus
7. Septic endocardial, valvular vegetations
8. Air embolism

side or have the patient cough; slowly irrigate the catheter as the balloon is inflated, and rotate the arm to a somewhat different position before repeating the wedging process. If no pressure monitor line fluctuation is seen, but a straight line is viewed on the oscilloscope, and electronic competency of the equipment is assured, the chances are excellent that the balloon is obstructing the tip of the catheter. The balloon should be deflated, the catheter withdrawn slightly, and the balloon reinflated with half of the volume of fluid previously used. Under any circumstance, the air should be removed from the balloon following the obtaining of the wedge pressure reading, as this will help to prevent further distal migration of the tip of the catheter.

Rupture of a branch of the pulmonary artery results in either *hemoptysis* or *pulmonary infiltrate*. Sudden, severe hemoptysis is invariably a result of such damage to the pulmonary arterial tree, and should be managed by turning the patient on the side in which the catheter resides, removing the catheter, and neutralization of any anticoagulants which may have been given. The hemoptysis will usually subside, although a pulmonary infiltrate may appear later. Persistent hemoptysis over several hours requires reinsertion of a catheter into the pulmonary artery, followed by injection of dye; if the site of rupture is visualized angiographically, it can be embolized with gel foam or other hemostatic pledgets in order to staunch the bleeding.

Arrhythmias occur with much greater frequency than with CVP catheters, since the balloon traversing the ventricle and pulmonary artery outflow tract stimulates the sensitive endocardium and valvular apparatus. These are to be expected, and are only of concern if ventricular tachycardia develops, in which case the catheter should be promptly removed rather than persisting in attempting to float the catheter tip into the pulmonary artery. Generally, when the catheter tip and balloon have progressed into the pulmonary artery proper, the arrhythmias subside and there is no further difficulty, unless the catheter tip is withdrawn from the pulmonary artery and resides in the right ventricle. Intravenous lidocaine may be given if premature ventricular contractions are observed.

Balloon rupture occurs on occasion, especially if the Swan-Ganz catheter is allowed to remain in place for longer than 3 days, or if repeated and frequent inflation of the balloon for monitoring of the wedge pressure is undertaken. Balloon rupture is easily detected by the inability to obtain a wedge, to reaspirate the fluid which has been infused, and, pathognomonic of this accident, aspiration of the balloon lumen yields blood. When this is observed, the catheter is customarily withdrawn slightly, and pulmonary artery end diastolic pressure substituted for pulmonary capillary wedge pressure, provided that the patient does not have ARDS. Practically, the catheter should be removed, since the rupture of the balloon allows for the potential embolization of bits of the latex rubber.

Knotting of the intracardiac catheter is usually a consequence of too forcible or too rapid introduction of the catheter. Once the Swan-Ganz catheter, with the balloon inflated, has reached the right atrium, it

should progress with very little, if any, manual pressure exerted to push the catheter tip forward. At this point, if there is any difficulty in introducing the catheter, obtaining a portable fluoroscopic unit or removal of the patient to the catheterization laboratory fluoroscopic unit should be considered. If the catheter does become knotted, the previously mentioned Caves-Shulz cardiac bioptome, a flexible, readily manipulable alligator jawed instrument, may be utilized to extract the knotted catheter or conceivably to untie the knot, under fluoroscopic control. Great caution should be exercised in removing a knotted catheter through the pulmonic or tricuspid valve, since valvular damage is likely to result.

Superior vena cava and *intracardiac thrombus*, or sterile vegatations, have been observed with some frequency in autopsied patients who have had indwelling catheters for several days or longer.[19, 28] Some of these give rise to pulmonary emboli and are undoubtedly due to endothelial and endocardial irritation by the catheter, including the to and fro motion of the catheter in assuming the wedge position and then being withdrawn from that position. In addition, several patients with *septic endocardial vegetations* have also been reported, with catheterization times as short as 5 days.[19] These usually develop in patients who have preexisting septic foci elsewhere, and in whom there is a nidus for bacteria to lodge on the damaged endocardium or valve leaflets of the right ventricle or atrium. Left heart vegetations are also reported with wedge pressure monitoring, obviously not directly due to the mechanical irritation of the catheter, but perhaps as a consequence of introduction of bacteria through the catheter tract in the skin and subsequently into the venous side of the central circulation.

Other complications of the Swan-Ganz catheter are similar to those of the CVP catheter, including broken embolized catheters, air embolism, and other complications (see Table 2). Additionally, a Swan-Ganz catheter in place in a patient undergoing open heart surgery is sometimes subject to damage or to being sutured in place during the course of intracardiac surgery (valve replacement, e.g.). This might not be noticed until an attempt was made to move the catheter, or to remove it after the operation. An elaborate way of removing such a catheter by first releasing it from the entrapping suture has been described,[6] but on several other occasions it has been necessary to resort to thoracotomy and removal of the catheter under direct vision.

Measuring *cardiac output* with the thermistor-tipped balloon flotation catheter has opened up an entirely new and extremely valuable monitoring modality in patients who are critically ill, or with profound alterations in cardiac output. On occasion, a central venous pressure catheter will be in place, and it will be necessary to insert a Swan-Ganz catheter in its stead. Rather than removing the central venous pressure catheter, a guide wire can be introduced via the CVP catheter; the CVP catheter is removed and the Swan-Ganz catheter introduced over the guide wire.[31] Although the possibility of introducing infection would certainly seem to be markedly augmented by such a technique, this did not prove to be the case in the series cited.

Major errors in cardiac output, however, can occur in two ways: first, the injectate may be allowed to rewarm prior to injection into the right atrial portion of the catheter, and it should be noted that holding the iced saline syringe in the operator's hand will introduce considerable error, as will allowing the syringe with iced saline to remain at room temperature for more than a few seconds. For that reason, when measuring cardiac outputs, it is imperative that the iced saline, removed from the water bath at 0° C, be used within 10 seconds, to minimize the error of allowing the temperature of the iced solution to rise. Second, the use of positive pressure ventilators with positive end expiratory pressure also introduces a considerable error, as noted previously, and accurate results require either allowing the patient to become apneic for a short period of time while the cardiac output is being monitored, or, at the very least, sampling should be done at precisely the same point in the expiratory cycle each time that the sample is drawn for cardiac output calculation.

ARTERIAL CANNULATION

Insertion of arterial catheters for blood gas sampling, pH measurement, and arterial pressure monitoring is a commonplace procedure when monitoring critically ill patients. The radial artery is most frequently selected for percutaneous cannulation, although the trend is to use the femoral or the superficial temporal artery in certain patients.[17, 37] The value of arterial pH and blood gases is incontrovertible, and direct measurement of intra-arterial pressure is much more accurate than use of the blood pressure cuff, particularly in patients with low peripheral arterial pressure. Since coronary flow is primarily dependent on diastolic pressure, it is desirable in hypotensive patients to provide a method for accurate recording of mean arterial and diastolic pressures.[5]

Indiscriminate radial artery catheterization has been subjected to considerable criticism recently, since untoward occurrences resulting in tissue loss in the hand have not been rare in the experience of those who monitor with this technique on frequent occasion.[4, 11, 14, 16] *Thrombosis* occurs in the radial artery in 20 to 40 per cent of patients in whom the radial arterial catheter is left in place for more than 12 to 18 hours.[4, 16] The incidence is higher if the patient is critically ill, is hypercoagulable, or has impaired tissue perfusion for some period of time, as with shock. Although not widely appreciated, patients may not have total occlusion of the radial artery, but may develop digital gangrene in one or two digits, secondary to *embolization* of pieces of intima, or of clot that forms on the radial artery catheter. A common digital vessel peripheral to the catheter insertion site is then obstructed. Such emboli, although much less common than arterial thrombosis, present an unpreventable cause of tissue loss in the hand.

Clotting of the radial artery which occurs with some frequency, especially in protracted radial artery catheterization, is usually innocuous, provided that the patient has reasonable tissue perfusion, and as-

suming that the ulnar artery is patent. This should always be demonstrated by a negative Allen test, done manually or with the Doppler apparatus prior to catheter insertion. Skin necrosis proximal to the site of radial artery cannulation has recently been reported, perhaps from spasm secondary to use of a large bore catheter.[7] Short, fine needles with a "butterfly" hub can suffice for intraoperative monitoring of arterial pressure and blood gases, and might prevent these sloughs. This type of needle must be removed at the end of operation, since any motion of the patient's wrist can cut or damage the radial arterial wall and create a serious periarterial hematoma.

The use of a femoral artery catheter is quite satisfactory, provided that the patient has a normal arterial tree, and is cooperative enough not to remove or dislodge the catheter inadvertently. In obese patients, or those with hypotension which makes ready palpation of the femoral artery difficult, attempts at percutaneous femoral artery catheterization may prove frustrating, if not damaging. If a surgical incision to expose the artery is obligatory for cannulation, then femoral artery catheterization should not be employed, but another site sought. The dorsalis pedis artery has been a site of election, again using a small needle with a butterfly flange or a very fine polytetrafluoroethylene catheter. This route has merit in a patient with adequate arterial perfusion pressure in whom the artery is readily palpable. Obviously, a strong posterior tibial pulse must also be palpable in order to assure adequate nutrition to the foot in the likely event that the dorsalis pedis artery will thrombose after a period of time.

The safest site for arterial cannulation has proven to be the superficial temporal artery.[4, 17] This vessel is approached through a short preauricular incision extending from the junction of the pinna of the ear with the scalp to the midpoint of the tragus, ordinarily 1.5 to 2.0 cm in length. With incision of the superficial fascia, the artery is easily dissected free of surrounding fat, and is ligated distally. A fine Silastic or other soft catheter is inserted into the artery for a distance of 3 to 4 cm, and a proximal securing ligature is placed around the catheter and artery. Using a three-way stopcock and a pressure infusion pump with dilute heparin solution to maintain patency, the catheters will function well for approximately 5 to 10 days, with a lesser incidence of catheter sepsis than those placed in the lower extremity, and no danger of tissue loss with thrombosis of the channel. Blood samples needed for any purpose are drawn because of convenience and excellent flow. Arterial pressure can be monitored in the intensive care unit, either continuously through one arm of the three-way stopcock or intermittently as desired.[4]

In the event of thrombosis of the radial artery with distal ischemia, early operation should be undertaken to assure patency of the radial artery and adequate blood flow to the hand. There is an unfortunate tendency to view the ischemic digits distal to such catheter insertion as having suffered temporary ischemia, and optimistically to rely on collateral circulation to maintain the tissue of the hand and fingers. Pain, coldness, and pallor of one or several digits after radial artery interrup-

tion is usually the prelude to tissue loss. The catheter should be removed, and a brachial angiogram done as an emergency. If the artery is occluded, and good ulnar flow to the hand is not seen, the arteriotomy should be exposed, and a Fogarty catheter gently inserted upward to the area of the elbow with clearing of all clot from the proximal radial artery. The use of a binocular loupe for arterial repair at the site of arterial catheter insertion is extremely helpful in a vessel of this size. The intima should be carefully inspected, any loose tissue resected or repaired, and the arteriotomy wound carefully closed with interrupted 7-0 polypropylene suture. In the majority of cases, adequate flow to the superficial palmar arch and to the digits can be reestablished, provided the delay between onset of symptoms and reconstruction is minimal.

There has been a tendency recently to introduce long catheters into the radial artery for purposes of *central arterial catheterization*. A 100 cm long Teflon catheter of a very fine caliber is inserted into the proximal arterial tree for arterial blood sampling and to monitor cardiac output by analysis of the aortic pulse wave.[16] The incidence of significant arterial insufficiency in the hand following this procedure was approximately 0.5 per cent, and three patients required thrombectomy for purposes of reestablishing effective arterial circulation. Sepsis can occur with arterial catheters which is essentially identical to the sepsis experienced with venous catheters. The management of these septic complications is the same as with venous catheters: removal of the catheter, meticulous culturing of the catheter tip, and administration of appropriate antibiotics.

Brachial and axillary artery puncture for monitoring or arteriography has resulted in similar complications, but also *compression neuropathy* has been seen, due to the formation of a large hematoma in the area of the brachial plexus with compression of the plexus.[12, 24-26] This particular complication has been noted primarily in patients on anticoagulants, but can also occur in patients with a presumably normal coagulation mechanism. The axillary artery is in close proximity to the brachial plexus, and cannulation of the axillary artery just distal to the humeral head, at the easiest point of access, may result in a large hematoma with plexus compression. The accumulation of a large quantity of blood under the fascia adjacent to the artery and neural structures is apt to cause paresis, hypesthesia, anesthesia, or severe pain in the hand due to compression by the clot. The patient who has had an arterial catheter inserted anywhere in an extremity vessel, closely followed by neurologic symptoms, should not be treated expectantly, since any distal neurologic symptoms make exploratory operation mandatory.[26] Nerve damage due to pressure may become permanent, with weakness and other neurologic findings, whereas early operation, within 12 hours of the arterial catheter placement, will relieve symptoms and allow complete functional return. In this light, considerable care with manual pressure applied to the arteriotomy site following the removal of the catheter is necessary, since the injudicious use of extreme pressure can, of and by itself, damage the adjacent neurostructures, particularly the median nerve in the upper limb.

As a basic rule, if pain persists for 2 hours or more after arterial puncture, surgical therapy may be necessary; if the pain in the distal extremity persists for 6 hours or more, the wound site must be explored, even if a large hematoma is not visible. Most often, there will be rapid accumulation of a large hematoma, with some dissection of blood into the distal part of the extremity. Under such circumstances, the dissection of blood in the subfascial plane has the same significance as crushing trauma, and Volkmann's ischemic contracture can result if prompt evacuation of the clot and liquid blood from the subfascial space is not undertaken. As with trauma, the suspicion that fasciotomy may be necessary should be sufficient reason to proceed with decompression of the subfascial structures, since an unnecessary decompression is unpleasant, but the failure to decompress, with subsequent loss of function, may result in the loss of a useful limb.

Nosocomial infections resulting from contamination of pressure monitoring devices have been largely ignored until very recently.[36] Bacteremia, fungemia, and serum hepatitis have been reported in patients who were monitored with transducers, pressure gauges, and strain gauges, when these were inadequately cleaned between uses. These instruments are in direct communication with the patient's intravascular space by cannulae and fluid conduits. Infection is not necessarily local, but may be manifested by distant foci of infection (endocarditis, mediastinitis) or by systemic sepsis, as with fungemias or serum hepatitis. Prevention is based on the following principles: Transducers should be cultured daily, flushing with small aliquots of infusion fluid, and inoculating both standard broth and anaerobic media; monitoring lines should also be cultured daily in the same fashion; blood should be prevented from entering the transducer if at all possible during the flushing process; tubing should be changed at least every 48 hours during monitoring; transducers must be disassembled, cleaned with strong soap, rinsed, and then sterilized after reassembly in ethylene oxide between patients. Although this is not a commonly recognized source of infection, it is apparently a very serious, although preventable, one.

COMPLICATIONS OF ANGIOGRAPHY DONE FOR EMERGENCY EVALUATION

Emergency angiography is occasionally necessary in patients with critical illness, including trauma, pulmonary embolism, acute arterial or venous occlusion, and in evaluation of visceral structure and function in various organs. Iodide containing dyes, such as diatrizoate and iothalamate, are sometimes used in very large doses in patients who require angiographic visualization of a large portion of the vascular tree, or in patients in whom repeated films are necessary to evaluate the course of a pathologic process.

Aside from frank iodide sensitivity, which can generally be detected by history or skin testing, the major problem with these dyes is the *nephrotoxicity* which accompanies their injection, especially in large

doses. It is well accepted that 10 per cent of patients who have angiographic studies show evidence of renal damage with the injection of the dye, although most of these have had evidence of preexisting renal or cardiovascular disease.[27] If the creatinine rises in the immediate postangiographic period, a plain abdominal film should be taken after 24 hours; if dye remains, outlining the kidney, there is poor excretion of the contrast, and this is presumptive evidence of significant renal damage. In patients who are dehydrated, who are known to have poor renal perfusion on the basis of preexisting renal disease or hypertension, in those with cardiovascular disease requiring treatment, with hyperuricemia, or with diabetes, there is a definite predisposition to renal injury when large amounts of these dyes are infused. The tolerance of dye in critically ill patients is such that 0.5 gm of dye per kg should not be exceeded; in extreme cases 1.0 gm per kg can usually be tolerated, but this certainly poses a very real risk of toxic nephropathy.

Prevention of these complications can be accomplished in many patients by proper hydration, ensuring that the patient has adequate extracellular fluid volume, prophylactic mannitol infusion (20 gm of mannitol as 100 ml of 20 per cent solution), and pre- and postangiographic monitoring of the urine output and creatinine.[30] In general, the more severely ill the patient, the greater the propensity for the manifestation of renal damage. In addition, the problem of *allergy* is essentially one of sensitivity to methylglucamine radical, a cation, which is thought to release histamine, cause bronchospasm, and diminish coronary perfusion. The prophylactic administration of an antihistamine does not seem to alter these allergic responses significantly, but support of the circulation in the event of an allergic reaction with dopamine, fluid loading, and mannitol is indicated.

SUMMARY

Although monitoring of critically ill patients has made giant steps forward in the past 15 years, such monitoring techniques must be viewed as a calculated risk, since a small but finite proportion of patients who are monitored by invasive techniques will suffer untoward and, occasionally, catastrophic consequences. Minimizing the risk involves careful general evaluation of the patient, adherence to strict indications for use of invasive techniques, and care coupled with experience in the actual manipulation. The specific procedure or procedures to be undertaken must be felt to be essential to the successful outcome of the care of the patient. The treatment of all critically ill patients must be individualized, and the monitoring techniques employed should be similarly individualized. With such a judicious approach, a great deal of valuable information can be obtained, and an effective and intelligent therapeutic regimen outlined so as to assure a successful outcome in as many patients as possible.

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Peptic Ulcer Surgery

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Peptic ulcer is one of the commonest diseases of the stomach and duodenum and will affect approximately 10 per cent of the population at one time or another.[41, 62] Surgical intervention is needed if medical treatment fails. Urgent or emergency operation may be necessary for perforation, obstruction, or bleeding. A variety of procedures for the treatment of ulcer disease has been advocated and these have altered the normal gastric physiology in various ways. At times, the complications following the surgery are more distressing to the patient than the original ulcer diathesis. It is the purpose of this article to review the diagnosis and management of these distressing postoperative problems (Fig. 1).

EARLY COMPLICATIONS

Hemorrhage

The reported incidence of intraluminal or intraperitoneal hemorrhage following gastric surgery is 1 to 3 per cent.[50] Intraperitoneal hemorrhage presents with signs of shock shortly after the operation without external evidence of blood loss. The bleeding is usually due to an unrecognized splenic injury or slippage of a ligature.[3] Other causes for persistent hypotension, such as cardiogenic shock, should be quickly ruled out. If prompt blood replacement of 3 to 4 units fails to correct the problem, reoperation and control of the bleeding site are mandatory.

Intraluminal bleeding generally presents as continued or intermittent bloody drainage from the nasogastric tube. The suture line is the usual source of the bleeding, but bleeding may occur from an unresected duodenal ulcer, gastric erosion, esophagogastric tear, or an overlooked gastric ulcer.[3, 50] In patients who have had a Hofmeister modification of the Billroth II gastric resection the usual source of bleeding is the closure of the lesser curvature. Initial management consists of iced

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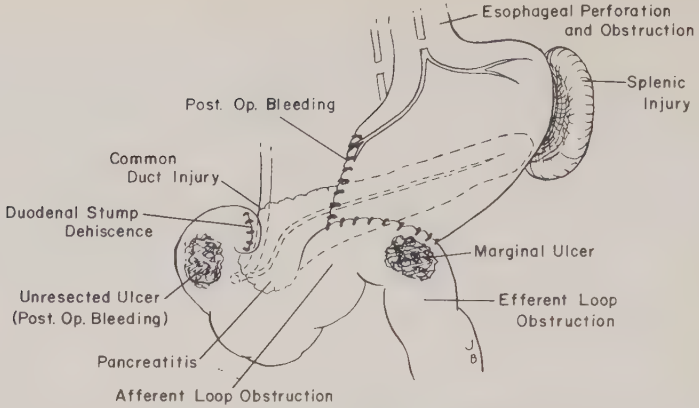

Figure 1. Some postoperative complications of peptic ulcer surgery.

saline lavage of the stomach, blood replacement and close monitoring of vital signs. An adequately functioning nasogastric tube is mandatory as a poorly functioning tube leads to distention of the gastric remnant with clots which predisposes to continued bleeding. A check of coagulation factors will rule out a bleeding diathesis. Consideration of selective arteriography with vasopressin infusion is appropriate if adequate facilities are available.[12] Approximately two-thirds of these patients will respond to conservative measures. If brisk bleeding continues requiring four to six transfusions to maintain vital signs, reoperation is indicated. Preferably, a longitudinal gastrotomy is made in the midline of the anterior wall of the gastric remnant, the suture lines everted into the wound, and a search for the bleeding point made (Fig. 2). Bleeding from the suture line should be oversewn. Other bleeding sites should be similarly managed.

When bleeding occurs between the seventh and tenth day postoperatively, it is usually from sloughing at the anastomotic line. This responds well to iced saline lavage and rarely requires reoperation. If bleeding

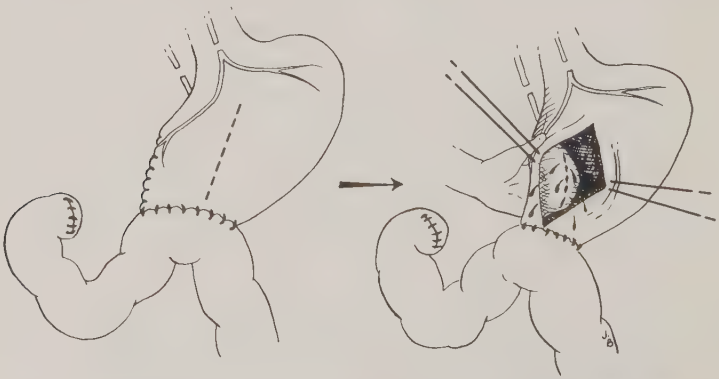

Figure 2. Obtaining control of suture line bleeding via a longitudinal gastrotomy and eversion of suture line into gastrotomy incision.

persists the possibility of another source of bleeding should be excluded by endoscopy.

Acute Gastric Dilatation

In the immediate postoperative period, distention of the stomach can produce epigastric pain, tachycardia, and hypotension. This picture can resemble a duodenal stump blowout, but is easily differentiated by the rapid improvement following insertion of a nasogastric tube or clearing of a plugged tube if one is in place.

The etiology of acute gastric dilatation is an altered breathing pattern in which the patient actually swallows inspired air. As the stomach becomes distended, gastric secretion is stimulated and fluid enters the lumen from the interstitial space. It is possible that up to 4 to 5 liters of fluid may accumulate, leading to dehydration, fall in plasma volume, and even death. Improvement in the clinical picture is dramatic and rapid following insertion of a nasogastric tube and restoration of plasma volume. The routine use of postoperative gastric decompression has minimized the occurrence of this unfortunate and preventable complication.

Pancreatitis

Postoperative pancreatitis is a rare but frequently fatal complication of gastric surgery. The mortality rate ranges from 30 to 50 per cent.[48, 63] Various etiologic factors have been implicated and it is probable that one or more may play a role. These include direct operative trauma to the gland or its ducts, impairment of the vascular supply, and stagnation of pancreatic and duodenal contents.[10, 48] These usually occur during mobilization of the duodenum from the pancreas or unwise efforts to dissect an ulcer from the pancreas rather than the stomach or duodenum from the ulcer bed.

Acute postoperative pancreatitis usually presents in the first 24 to 48 hours postoperatively with increased abdominal pain, direct and rebound tenderness, hypotension, tachycardia, leukocytosis, and hyperamylasemia. Unfortunately, these same signs, symptoms, and laboratory findings may occur after duodenal stump or anastomotic leaks. An x-ray of the upper gastrointestinal tract with a water-soluble material may differentiate the conditions. If a presumptive diagnosis of pancreatitis is made, treatment consists of nasogastric suction, analgesics, antibiotics, and vigorous fluid replacement. If a definitive diagnosis cannot be made and the patient continues to deteriorate, reoperation is indicated to rule out another life-threatening intraabdominal condition such as anastomotic leak, intestinal obstruction with strangulation, acute cholecystitis, or acute afferent loop syndrome.[16] If acute pancreatitis is found on reexploration, drains should be placed about the pancreas and consideration given to the insertion of peritoneal catheters for dialysis.[4] Total parenteral nutrition may be necessary for adequate nutrition during this severe catabolic state in addition to the vigorous supportive measures described above. If pancreatic abscess should develop, reexploration with adequate drainage is mandatory.

Anastomotic or Duodenal Stump Dehiscence

An anastomotic or duodenal stump leak is a potentially lethal complication. Anastomotic disruption is due to a technical error, poor general nutrition with hypoproteinemia, or rarely, excessive devascularization of the stomach remnant.[49] Duodenal stump leakage is secondary to insecure closure of the duodenal stump whether due to a technical error, edema or scarring of the duodenum, pancreatic injury with a local pancreatitis, duodenal ischemia, or duodenal distention from proximal loop obstruction.[38] Early recognition of a difficult duodenal dissection will allow the surgeon to select an alternate operation such as vagotomy and gastrojejunostomy. If a gastric resection is performed and duodenal closure is difficult, consideration should be given to tube duodenostomy in an effort to prevent duodenal stump leakage.

The clinical presentation of either complication is similar and may also be confused with acute postoperative pancreatitis. Acute pancreatitis, however, generally occurs in the first 24 to 48 hours postoperatively, whereas suture line dehiscence usually presents 3 to 7 days after operation. Typically, the patient is recovering uneventfully and then suddenly develops severe abdominal pain with rigidity, fever, increased white blood cell count, and frequently, hypotension. An x-ray study with a water-soluble substance may occasionally be necessary to confirm the suspected leak.

After immediate resuscitation including nasogastric suction, antibiotics, and appropriate parenteral fluids and electrolytes, definitive management demands adequate surgical drainage. Through an appropriate incision a sump drain is placed near the site of leakage. Continuous suction must be maintained until an adequate tract is established. Maintenance of adequate nutrition is essential if these patients are to recover and heal their wounds. Total parenteral nutrition has proved to be a particularly effective adjunct. Gastrointestinal secretions are decreased while on total intravenous nutrition which aids in fistula closure by decreasing the volume of the discharge.[17, 23, 65] An effective alternative is the use of a feeding jejunostomy distal to the site of leakage. As long as nutrition is maintained and sepsis controlled, most of these fistulas will close spontaneously in 3 to 4 weeks.

Jaundice

Jaundice following gastric surgery is an alarming situation and one of great concern for the surgeon. Postoperative jaundice may follow any major surgical procedure, but it occurs more frequently after biliary tract and gastric surgery. Of particular concern to the surgeon is the possibility of common bile duct injury during the gastric resection.

In addition to common bile duct injury postoperative jaundice often has a multifactorial etiology including shock, sepsis, hepatotoxic drugs, anesthetic agents, multiple transfusions, renal failure, and preexisting liver disease.[44, 45] A complete diagnostic workup is essential prior to any consideration of operative intervention in order to clearly differentiate hepatocellular dysfunction from extrahepatic obstructive jaundice.

A systematic approach for the evaluation of jaundice is mandatory. First, all anesthetic agents, medications, and the number of transfusions should be reviewed. The evolution of the jaundice should be carefully monitored by serial liver function tests including serum bilirubin, alkaline phosphatase, and serum glutamic oxaloacetic transaminase.[45] A search for sepsis, identification of hemolysis, and evaluation of renal function will further clarify the source of the jaundice in most cases.[44]

The majority of patients who develop postoperative jaundice have a nonmechanical basis for their jaundice. Persistence of jaundice after 3 to 4 weeks suggests a mechanical cause and further diagnostic procedures must be instituted to determine the etiology. The most useful procedure is percutaneous transhepatic cholangiography.[20] If diagnostic workup reveals injury to the common bile duct, operative repair is necessary.

During difficult duodenal dissections, common duct injury may be prevented by choledochotomy and insertion of a probe for duct identification. This will permit safer management of the duodenal stump closure. As previously emphasized, early recognition of a difficult duodenal dissection and potential common bile duct injury will permit the surgeon to select an alternate operation such as vagotomy and gastrojejunostomy.

Delayed Gastric Emptying

Some delay in gastric emptying is often present following gastric surgery. However, this usually resolves within 1 week. At times, this failure to empty may persist for a longer time on either a mechanical or functional basis and lead to vomiting with electrolyte imbalance.

The mechanical causes may be secondary to edema at the anastomosis, adhesions obstructing the distal loop, intussusception of the jejunum, or internal hernia. The edema will resolve with continued nasogastric suction and proper maintenance of fluids, electrolytes, and nutrition. The other etiologies usually present as high small bowel obstruction requiring laparotomy and appropriate operative correction as dictated by the findings at reexploration.

There is another group of patients whose failure to empty is on a nonmechanical basis. The obstruction cannot be considered functional until a complete diagnostic workup has ruled out an organic etiology. Metabolic causes as hypokalemia, hypoproteinemia, or hyponatremia must be considered and corrected if they exist. An upper gastrointestinal x-ray will merely confirm the suspicion that the stomach fails to empty. Fiberoptic endoscopy is the most important diagnostic test in ruling out an organic cause for gastric retention. If delayed emptying persists in spite of normal chemistries and the absence of mechanical obstruction, prolonged nasogastric suction and total parenteral nutrition should be instituted. These measures may be necessary for up to 4 to 6 weeks with intermittent attempts at feeding (preferably solids such as hamburger) in an attempt to stimulate effective peristalsis. Reexplora-

tion should be reserved as a last resort and rarely contributes significantly to the solution of the problem.

Recently, an investigational drug, metoclopramide, has been used with good results.[24, 43] This drug has the net effect of accelerating gastric emptying and small bowel transit. No action on gastric acid secretion has been noted. In our experience this pharmacologic adjunct shows promise in the management of these discouraging and frustrating problems related to postoperative gastric stasis.

Postoperative gastric stasis is almost nonexistent after parietal cell vagotomy, the newest of the peptic ulcer operations. Preservation of antral vagal innervation permits unimpaired emptying of solid food in the absence of pyloric or duodenal stenosis.[32, 64]

Infection

Following any intraabdominal operation, septic complications may occur. However, the patient with obstructing or bleeding duodenal ulcer is at greater risk than one undergoing elective laparotomy for peptic ulcer disease. Nichols[46] has shown that the stomach is colonized in these patients and suggests a possible role for prophylactic antibiotics. Snyder[58] reported a wound infection rate of 10 per cent after operations for massive bleeding, whereas an infection rate of approximately 3 per cent is found in most series of elective gastric surgery.[38] The wound infection typically appears on the third to fifth postoperative day with fever, incisional pain, tenderness, and wound erythema. The wound should be opened and appropriate cultures taken. Saline dressings should be applied and the wound allowed to close by secondary intention.

Intraabdominal infection may present as diffuse peritonitis or as an abscess anywhere within the abdomen. Diffuse peritonitis is usually a catastrophic event associated with a disrupted anastomosis or leaking duodenal stump. The diagnosis and management of these complications is discussed elsewhere in this review.

The diagnosis of intraabdominal abscess may be extremely difficult. The patient will usually have general malaise, anorexia, low grade temperature, and varying degrees of leukocytosis. Every effort must be made to localize and confirm the presence of an abscess. Plain films of the abdomen may show an extraluminal collection of gas with an air-fluid level. Chest x-rays may demonstrate a pleural reaction secondary to a subphrenic abscess. A combined liver-lung scan may be beneficial. Recently, gallium scans combined with abdominal echograms have been useful in localizing intraabdominal abscesses.[36] Once localized, adequate surgical drainage is the treatment of choice.

Esophageal Complications of Vagotomy

The complications specifically due to vagotomy are difficult to distinguish from those effects related to a concomitant drainage procedure or gastric resection. Esophageal perforation, bleeding, postvagotomy dysphagia, and periesophageal fibrosis account for 5 per cent of the immediate postoperative complications and are particularly related to the technical aspects of vagotomy.[51, 66]

Perforation may occur during mobilization and dissection of the lower portion of the esophagus. Recognition and closure with interrupted nonabsorbable sutures almost always results in satisfactory healing. If unrecognized, however, the potentially lethal complications of fistula, sepsis, subphrenic abscess, peritonitis, and mediastinitis may ensue.

During vagotomy hemorrhage may occur from splenic injury or bleeding from periesophageal vessels. This latter complication can be prevented by ligation or application of silver clips to the ends of the divided vagus nerves.

Postvagotomy dysphagia is usually noted seven to 14 days postoperatively and is probably due to direct injury of the esophageal wall at the time of vagotomy.[47] It usually resolves spontaneously in 3 weeks to 3 months. Persistence of symptoms is corrected by one or two esophageal dilatations. Periesophageal fibrosis, however, usually presents as dysphagia approximately 3 weeks postoperatively and may progress to complete obstruction. This complication should be suspected if esophageal dilatation is ineffective in providing lasting benefit. Excision of the dense scar tissue about the esophagus, preferably via thoracotomy, is necessary for relief.

Complications peculiar to selective and parietal cell vagotomy are operative injury to the lesser curvature or necrosis of the stomach from devascularization.[33] If recognized, perforations should be closed. Unrecognized perforations or sloughing of the lesser curvature of the stomach will result in serious complications such as gastric fistula, sepsis, abscess, or peritonitis. Care in dissection along the lesser curvature will minimize these potentially lethal complications.

LATE COMPLICATIONS

Afferent and Efferent Loop Obstructions

Early obstruction of the afferent loop may contribute to duodenal stump leakage. However, in the typical "afferent loop syndrome" the patient experiences upper abdominal pain within a few minutes after eating. Further confirmatory evidence may be the presence of a palpable mass. Vomiting of bile will produce relief with disappearance of the mass. Elevations of serum amylase and jaundice can occur. If the diagnosis is suspected, it may be demonstrated with upper gastrointestinal contrast studies. Endoscopy should be performed in all patients in whom the diagnosis of afferent loop obstruction is suspected. Its main value is to rule out other causes of the patient's symptoms, especially alkaline reflux gastritis.[8, 9] This complication is best avoided by the use of a short afferent loop and care in fashioning the anastomosis to prevent obstruction at the stoma. If the problem does occur, surgical intervention is indicated and may require mere lysis of adhesions (rarely), jejunojejunostomy (Roux-en-Y or side-to-side), or complete revision of the anastomosis. If a vagotomy had not been performed with the

first operation, it should be added if the duodenal contents are diverted distal to the gastrojejunal anastomosis.

Efferent loop obstruction secondary to adhesions usually presents as a small bowel obstruction and is treated accordingly. Internal hernias most often follow the use of an antecolic anastomosis with herniation between the anastomosis and the transverse colon.[59] Once recognized, laparotomy is indicated with reduction of the hernia and closure of the defect. Jejunogastric intussusception presents as a high small bowel obstruction or upper gastrointestinal bleeding.[60] A mass can often be felt in the epigastrium. Emergency operative intervention and reduction is necessary. Any gangrenous bowel is resected. After reduction, nothing further is needed as most do not recur.

Alkaline Reflux Gastritis

Alkaline reflux gastritis is being recognized more frequently as a sequel of gastric surgery[61] in which the pylorus is bypassed, removed, or rendered incompetent. This has been reported after pyloroplasty, gastric resection with gastroduodenostomy (Billroth I) and gastrojejunostomy with or without gastric resection.[5, 25] An associated vagotomy has usually been performed.

The pathophysiology of the syndrome is not yet completely elucidated but the reflux of duodenal contents including bile acids plays a major role. Injury to the gastric mucosal barrier by bile acids may allow back diffusion of hydrogen ions and further contribute to the pathology.[31]

Clinically, the syndrome presents as persistent pain accentuated after meals often associated with nausea and vomiting. Weight loss may occur as the patient avoids food in order to minimize the pain. It is difficult and often impossible to differentiate this problem from the afferent loop syndrome on the basis of history. The most pertinent distinguishing features relate to the persistence of the epigastric "burning" with reflux gastritis only partially relieved by vomiting in contrast to complete arrest of symptoms after vomiting in intermittent afferent loop obstruction.

Once the diagnosis is suspected, work-up includes gastric analysis, barium upper gastrointestinal studies and endoscopy. Gastric analysis will show little or no acid, but complete collections are difficult and specimens generally contain bile. Barium studies may show a dilated afferent loop or another unexpected anatomic abnormality. The most important diagnostic test is direct visualization and biopsy of the gastric mucosa by endoscopy. A diffuse gastritis manifested by congestion, easy bleeding, adherent mucous and punctate erosions with free reflux of bile is seen gastroscopically.[5] Biopsy shows decreased parietal and chief cells with a predominantly lymphocytic inflammatory response. The mucosa is ulcerated, atrophic, and the gastric glands have a corkscrew appearance.[7] Paradoxically endoscopic signs of severe gastritis may be seen in patients with minimal or no symptoms.

Mild cases have been occasionally managed successfully with diet, antispasmodics, and cholestyramine. The reports concerning cholestyra-

mine are conflicting. Eckstam[18] observed a good response in 50 per cent of treated patients but Herrington[25] noted a poor response in 10 patients. Most investigators have reported poor success rates with all forms of conservative treatment for reflux gastritis. Recently an investigational drug, metoclopramide, has been tried.[14]

If conservative measures fail, surgical intervention is often beneficial. Diversion of duodenal contents from the gastric remnant by a Roux-en-Y revision is the preferred operation[25, 61] (Fig. 3). Repeat gastric biopsies have shown marked improvement and some have even reverted to normal.[8]

It should be emphasized that not all patients with pain and vomiting after gastric surgery have reflux gastritis and the combination of symptoms described plus characteristic endoscopic findings is essential for the diagnosis.

Dumping Syndrome

Dumping syndrome refers to a symptom complex that occurs in approximately 10 per cent of patients after gastric surgery.[42] It can be divided into early and late phases depending on the time of occurrence after eating.

The late symptoms are due to a reactive hypoglycemia which characteristically occurs 1 to 4 hours after a meal.[57] Central nervous system symptoms are most prominent but weakness, faintness and diaphoresis may occur. In contrast to the early phase, gastrointestinal symptoms are absent. This late syndrome can be controlled with a low carbohydrate, high protein, high fat diet, and only rarely is an intractable case found.

Early postprandial dumping is the more severe and disabling condition. Ten to 15 minutes following a meal the patient develops epigastric fullness, flushing, sweating, tachycardia, diarrhea, and occasionally transient hypotension. At times the attacks are so severe that the patient

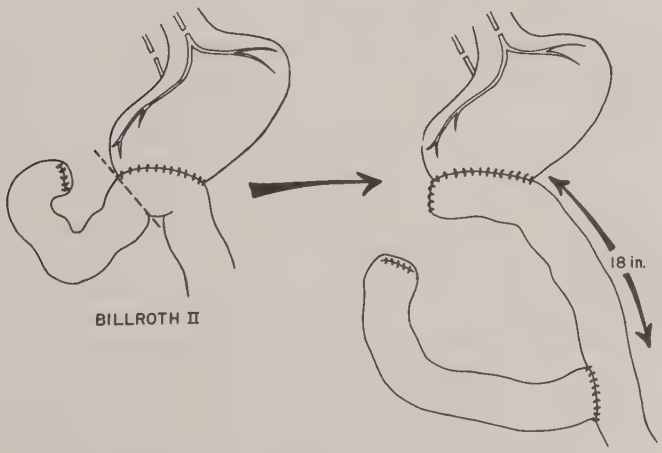

Figure 3. Roux-en-Y diversion of duodenal contents.

avoids eating and malnutrition and weight loss result. The pathogenesis is still not completely understood, but is undoubtedly due to a combination of factors. There is rapid emptying of a high osmolar load into the jejunum. This results in extracellular fluid shifts and a fall in plasma volume.[26] More recently implicated are the effects of a number of humoral agents including serotonin and the kinins.[13, 39]

The preferred management of these patients is medical with multiple small feedings low in carbohydrate, avoiding liquids while eating and lying down after meals. Various pharmacologic agents have been tried but have not been helpful. Surgical intervention should only be considered if severe symptoms accompanied by malnutrition persist after careful and prolonged medical management. Inconsistent improvement has followed a variety of remedial operations. Suggested procedures include conversion of a Billroth II gastrojejunostomy to a Billroth I gastroduodenostomy or the interposition of an antiperistaltic jejunal limb between the gastric remnant and upper jejunum.[52] As these procedures have an ulcerogenic potential, a vagotomy should be added if not performed at the first operation.

As with any complication, prevention is the best treatment. The incidence of dumping is reported to be minimal following parietal cell vagotomy.[27, 34, 54] However, before advocating this procedure for its lack of side effects, it must stand the test of time for effectiveness in controlling the ulcer diathesis.

Recurrent Ulcer

Recurrent or anastomotic ulceration is a serious complication of peptic ulcer surgery. The patient presents with recurrent pain, typically to the left of the midline, which is usually more severe and persistent than the pain of the original ulcer.

Diagnosis of an anastomotic ulcer by barium studies is frequently difficult. Fiberoptic endoscopy is the more accurate method for establishing its presence. Once demonstrated, the probable cause must be investigated as this will dictate subsequent treatment.

The causes of anastomotic ulcer include incomplete vagotomy, inadequate gastric resection, retained antrum, and Zollinger-Ellison syndrome. A Hollander test and/or basal and maximal secretory tests should be performed in order to determine the adequacy of vagotomy.[55] However, these are often difficult to interpret due to incomplete collections or contamination of the specimens with bile. Achlorhydria or very low secretory rates suggests a diagnosis other than recurrent ulcer. Serum gastrin levels are helpful in the diagnosis of Zollinger-Ellison syndrome. If the basal serum gastrin levels are normal, a calcium infusion test should be performed as some patients with a gastrinoma have normal basal serum gastrin levels but show a marked increase with calcium stimulation.[28, 30] Repeated serum calcium levels are helpful in ruling out hyperparathyroidism as a cause of recurrent ulceration.[2] If hyperparathyroidism is diagnosed, consideration should be given to parathyroidectomy prior to a direct surgical attack of the ulcer, as correction of the hypercalcemia may reduce the gastric acid secretory rate. The possibil-

ity of retained gastric antrum must be considered. If available, a technetium pertechnetate scan[11] or secretin infusion test[28, 29] may be helpful in differentiating retained antrum from gastrinoma. Demonstration of gastric mucosa in endoscopic biopsies obtained from the proximal end of the afferent loop is definitive evidence for retained antrum.

Treatment depends on the cause. If vagotomy is incomplete, transthoracic vagotomy is appropriate if subsequent healing of the ulcer will not result in gastric outlet stenosis. If vagotomy is complete, antral resection should be done. Further resection and vagotomy (if not done or incomplete) may be necessary in previously gastrectomized patients. If a Zollinger-Ellison syndrome is diagnosed, a total gastrectomy will be needed.

When dealing with recurrent ulcer, the surgeon must consider the initial operation and the reason for recurrence before selecting a corrective operative procedure.

Metabolic Disorders

Following gastric surgery bone disease, anemia, malnutrition, weight loss, and chronic diarrhea are known late complications.

Both osteomalacia and osteoporosis can occur after gastric resection. The etiology may be related not only to possible calcium loss in the stools but also reduced oral intake of food and calcium.[19] The treatment consists of appropriate doses of oral calcium and vitamin D.

Postgastrectomy anemia may result from inadequate stores of folic acid, iron, and vitamin B_{12}.[37] Deficiencies of one may mask deficiencies of the other. A complete hematologic evaluation is necessary to sort out the relative effects of each. A detailed discussion of these anemias is beyond the scope of this review, but their existence should be ascertained and appropriate medical measures taken to correct them.[56]

Malnutrition and chronic weight loss are usually secondary to inadequate intake and/or malabsorption. Malabsorption may be related to decreased acid production, pancreatic exocrine insufficiency, or bacterial overgrowth.[35] Oral pancreatic extracts will correct pancreatic exocrine insufficiency. Postoperative cultures have demonstrated significant colonization in the small bowel after the traditional peptic ulcer operations.[15, 21] The physiologic significance of this observation is not yet clear.

Chronic diarrhea following peptic ulcer surgery can be a particularly annoying problem. The etiology of "postvagotomy diarrhea" is unknown, but on occasion may be secondary to steatorrhea.[6] If the diarrhea is incapacitating and persists beyond 3 months, possible etiologies should be investigated. Such conditions as lactase deficiency bacteriologic or parasitic etiologies, sprue, and IgA deficiency should be ruled out.[22, 40] These patients should be treated with meals of low fluid content, antidiarrheals, pancreatic extracts if necessary and possibly cholestyramine.[1] Surgical intervention should be reserved for only the most intractable case. An antiperistaltic jejunal segment interposed 100 cm distal to the ligament of Treitz has met with limited success.[53] If parietal cell vagotomy proves to have a low recurrent ulcer rate, it may obviate the postvagotomy diarrhea syndrome.

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Breast Surgery

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The primary consideration in surgery of the breast centers about its most important disease, cancer. Efforts at early diagnosis, using axillary node involvement as the criterion, are effective at an earlier stage. Once suspected, biopsy confirmation of cancer is necessary prior to instituting therapy. Diagnostic errors may be encountered. The dispute over choice of primary treatment continues, with little hope of early agreement. The treatment selected may itself cause increased incidence of recurrent disease.[9] Complications may follow any diagnostic maneuver or method of therapy. Erors and complications are reviewed and measures for avoiding them suggested.

ERRORS IN DIAGNOSIS

Disregarding Symptoms

The earliest source of error is to disregard symptoms, particularly in the high risk group of patients. Every symptomatic patient deserves a baseline mammogram,[16] even if a palpable lesion is present and biopsy is to be done. The purpose of mammography is to discover clinically occult lesions lurking in the same or opposite breast. An additional potential advantage of mammography is the identification of a type of dysplastic change in which there is reported a markedly increased incidence of carcinoma;[40] this information may help define the high risk group more precisely. A false negative interpretation of the mammogram may also delay the diagnosis. Any breast symptom, finding, or mammographically detected lesion must be carefully evaluated and carcinoma excluded.

Benign Appearing Lesions

Carcinoma mimics a variety of benign conditions. Lesions which appear to present inflammatory processes may also cause diagnostic errors. Persistent nipple eczema, peripheral abscesses, and recurrent

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particular duct lesions[7] all require surgical therapy and tissue should be submitted for microscopic study. Bloody nipple discharge is an indication for duct excision, and, while usually due to a benign intraductal papilloma, intraductal carcinoma must be excluded. Any tissue removed from the breast requires thorough examination of its nature.[1, 2]

The Nonpalpable Lesion

Lesions found only on mammographs must be difficult to find at operation. A mammographic map is constructed using coordinates from the bi-plane mammograms. The relative position of the lesion in the breast is plotted after simulation of the mammographic position.[1, 2] Radiopaque markers on the skin may be helpful for orientation. Excision of a wide area about the calculated location of the lesion is then performed. This method of localization is also the best way in the most unusual procedures described below.

Utilizing mammographic control, the lesion may be prepared for operative localization by any of the following:

a. Insert one or more straight needles into or adjacent to the lesion and leave in place for operative orientation.[3]
b. Leave the tip of the thumb sheath of an extension type needle in the region of the lesion and tie the sheath to the skin by suture.[4]
c. Impale the lesion with a harpoon-like barbed wire wire.[5]
d. Inject a mixture of radiographic dye and methylene blue or comparable radiographic and visual markers.[6]

This latter "tool" technique is the one we use with satisfactory results.

Specimen mammographs must be performed during the operation to assure removal of the suspect lesion.

The nonpalpable lesion must not be visible in the biopsy specimen. If the lesion is not found on specimen mammograph, further excision and radiographic confirmation are performed until it is identified. When the lesion is present, the specimen may be sliced with radiographs of the sectioned pieces performed to pinpoint the lesion for the pathologist.[1, 2]

Palpable Lesions

The cause of any palpable lesion must be determined. Lesions that appear cystic and are mammographically negative may be aspirated.[8] If the mass disappears and the aspirated fluid is not bloody observation is appropriate. Cytologic study of the fluid is infrequently performed and is rarely of diagnostic value. Follow-up physical examination at about 6 weeks and again in 3 to 4 months is suggested, since early recurrence of the mass may be due to the presence of a neoplasm. Any tumors which reappearing in the same location are excised for biopsy.

If aspiration produces bloody fluid, or if any portion of the original mass remains on review, arrangements should be made for early excision biopsy using the previous wound to localize the area to be biopsied.

Palpable lesions which are not cystic are usually excised. The

preferred incision is circumareolar whenever the lesion can be reached by that route. Peripheral lesions are approached by a curvilinear incision in Langer's lines. General anesthesia is used more frequently. Local anesthesia is satisfactory for excision of many lesions. Hospital admission and costs are reduced. Deep lesions and those that are not palpable should be excised under general anesthesia.

Access to multiple lesions may be obtained by means of a single incision in the inframammary fold, elevation of the breast tissue off the pectoralis fascia, eversion of the breast, and removal of the lesions. Small lesions may be difficult to identify by this approach; a wire suture with barbed end placed through a needle and fixed into the lesion is useful in localizing the lesion. While drains are avoided for most biopsy procedures, this approach involves considerable dissection, and suction catheter drainage is recommended.

Sample Biopsies

Removal of a portion of a lesion for study has merit as it is a direct approach with minimal disturbance of surrounding tissue. A positive biopsy diagnosis is reliable. Errors result from negative reports, since one cannot be certain that the specimen taken is representative. The biopsy may have been from an area adjacent to the tumor, from reaction about the tumor, or from a benign lesion which may also harbor a malignancy. A negative sample biopsy should be followed by excision and study of the entire mass.

Incision and needle biopsies are familiar procedures which provide tissue for diagnosis. Fine needle aspiration is a more recent procedure which is used increasingly and depends upon cytologic study. It is often confused with and should be differentiated from both cyst aspiration and needle biopsy (which obtains a core of tissue). Fine needle aspiration is used for palpable or with mammographic control for nonpalpable lesions. The liquid aspirate is smeared and stained; a high degree of accuracy is achieved by experienced aspirators and cytologists. This procedure is suggested as a way to increase the rate of positive surgical biopsies from the generally accepted 20 to 30 per cent. Knowing the diagnosis with a high degree of confidence, preoperative preparations and operating room scheduling are facilitated. The occurrence of false negatives (5 to 15 per cent) and the possibility of even a rare false positive (up to 2 per cent) justifies a formal biopsy prior to definitive treatment.

A rare source of error is the assumption that a carcinoma in the breast is necessarily a carcinoma of the breast. A metastatic lesion to the breast may be evident before recognition of the distant primary. The possibility of multiple primary neoplasms may also mislead the surgeon into assuming that the metastasis is a primary tumor. The type of tissue may give a clue that the tumor is metastatic. Diagnosis of lymphosarcoma or leukemic deposits will not affect therapy. Lack of recognition that a carcinoma is metastatic may lead to inappropriate therapy directed toward breast cancer.

CHOICE OF PRIMARY OPERATIVE PROCEDURE

Cancer is a serious disease; the possibility of cure should not be compromised by less important considerations. The extent of therapy is reasonably limited by its resultant morbidity and effect on functional status. Purely cosmetic considerations must be subordinated to the primary goal: the chance for a cure. Within limits, the procedure may be adapted to the type of tumor and extent of disease.

Many procedures and combinations of modalities of therapy are advocated, with data to support any view. The surgeon's decision is based on his evaluation of the present state of knowledge. He must communicate the reasons for his decision to the patient.

For the patient with a proven carcinoma of the breast, who has either no evidence of metastatic disease or none beyond the ipsilateral axilla, treatment is based on the following assumptions:

a. Breast cancer is multicentric.[25, 31, 35] It is an error to leave a second focus of cancer in the operated breast, whether that lesion is itself primary or metastatic from the known lesion. Subcutaneous mastectomy does not remove all breast tissue.[14, 41] Cancer may develop later in residual breast tissue.
b. Accessible lymph nodes which contain cancer should be removed. If the axilla is the only site of spread, opportunity for cure is enhanced.[18] The most reliable prognostic information comes from knowledge of axillary lymph node status.[38]
c. Axillary lymph nodes left in place do not augment the immunologic status of the host.[22, 34]
d. The wider the excision, the less the chance of cutting across involved tissue.[9]

The procedure used for most of our patients is best described as a total mastectomy with complete axillary dissection. Complete axillary dissection requires that the pectoralis minor muscle be liberated in some manner. Standard radical mastectomy[15, 17] is reserved for patients who have larger lesions (over 3 cm), involvement clinically or mammographically of the pectoralis fascia or muscles, or enlarged axillary nodes. Radiation therapy is added for operable patients with large medial hemisphere tumors. We do not employ the extended radical mastectomy.

Modified radical mastectomy techniques appear to: (a) satisfy the requirements of adequate cancer surgery, (b) give survival results equal to that of the standard radical mastectomy, and (c) alleviate concerns about disfigurement and late arm edema. The results of axillary node counts and patient survival[26, 27, 29, 33] lend support for its use. However, there is evidence that such axillary dissection leaves lymph nodes which are usually removed by standard radical mastectomy.[21]

The diagnosis of lobular carcinoma in situ requires at least a total mastectomy. The procedure is readily extended to include the lower axillary nodes (lateral to pectoralis minor) with only slight increase in operating time or morbidity.

THE OPERATION

The incision we use for the total mastectomy with complete axillary dissection is a transverse one which curves up laterally into the lower axilla. Planned standard radical mastectomy is performed by a vertical incision. The arrow-head (Greenough) incision is well adapted for upper outer quadrant lesions in which wide excision might not preserve sufficient flap to cover the axillary structures. The flaps for any of the variations of mastectomy are fashioned in the same manner. Thin skin flaps are formed, with gradual increase in thickness as the dissection proceeds. Minimal bleeding from the flap indicates that the dissection is at the proper level.

Removal of the breast with pectoralis fascia starts medially and continues laterally toward the axilla. The deep projections of pectoral fascia and breast tissue extending between fibers of pectoralis major muscle are visualized by upward traction on the specimen and excised. The dissection in continuity frees the interpectoral tissue and the lower axillary contents to the lateral border of the pectoralis minor. The exposure is facilitated by elevating the previously draped arm to relax the pectoral muscles and permit their retraction medially. All tissue below the axillary vein and medial to the latissimus dorsi tendon is included. This is the extent of the procedure of total mastectomy and partial (lower) axillary dissection. Obviously, the apical axillary nodes are not removed if the pectoralis minor is undisturbed. This is also referred to as a modified radical operation.[2, 7, 24, 27] The terminology is confusing, and should be based on description of extent of the axillary dissection.

Total mastectomy with complete axillary dissection requires that the pectoralis minor, which obstructs the passage to the apical area, be dealt with. The neurovascular supply to the retained pectoralis major muscle should be spared. One technique splits the pectoralis major over the level of the axillary apex, gaining exposure to the apical tissues; the area medial to the pectoralis minor is well visualized. The medial dissection communicates with that of the lateral approach. Another method is that of Patey[20] which resects the pectoralis minor, sparing the lateral pectoral nerve. Yet another approach divides the pectoralis minor at its coracoid insertion, permitting it to contract caudad; the lateral pectoral nerve is pulled down into the area of the dissection.

The method we employ to resolve this problem is to divide the origin of the pectoralis minor from the chest wall. This allows the muscle to contract cephalad and helps retract the lateral anterior pectoral nerve out of the field. The exposure is satisfactory and the number of axillary nodes removed equals that of the radical mastectomy.[33]

Hemostasis is accomplished by cautery for most of the procedure. Fine nonabsorbable suture material is used for ligating the perforating vessels, as well as the branches of the axillary vein and the vessels crossing it. Suture ligation is useful for muscular vessels and retracted bleeders. Precise hemostasis is required; only the vessel and minimal

surrounding tissue should be grasped with the hemostatic forceps. Visible vessels are clamped before division.

The flaps are handled gently and protected by moist sponges. Compression and pressure from instruments are avoided. Prior to closure the flaps are elevated and inspected for any further bleeding. Irrigation with saline keeps the wound from becoming dehydrated, flushes out clot and debris, and aids in identifying bleeders.

The widely dissected flaps of any type of mastectomy lead to empty ("dead") space and fluid accumulation. Stents or deep sutures are used to tack flaps down to the chest wall and reduce this potential "dead" space. Suction catheter drains are the most effective in removing blood and serum and in collapsing the flaps against the thoracic wall. Irrigation during closure also helps keep the suction catheters from becoming plugged too early.

Closure of the skin is by interrupted sutures. Tape closure can be used, but the movement of the thorax and moisture from the wound may shift the tapes and cause separation of wound edges before they adhere to underlying structures. Tension is avoided. The flaps can be further dissected laterally beyond the latissimus dorsi, medially across the midline, or inferiorly to provide additional mobility.

Skin graft is used for closure in about one-third of our cases. Split-thickness skin is taken from the lateral aspect of the opposite thigh using a separate sterile setup. The graft is sutured to the flap edges. On occasion skin may be dissected from the inferior margin of the specimen to provide a full-thickness graft.[3]

COMPLICATIONS OF MASTECTOMY[16, 28]

Preoperative Complications

Biochemical values suggestive of possible metastatic disease should be pursued, particularly elevations of the serum calcium and alkaline phosphatase. Carcinoembryonic antigen levels are occasionally elevated in breast cancer;[36] a baseline determination is valuable for comparison if subsequent changes occur. Arrangements should be made for Estrogen Receptor determinations on the fresh primary tumor.[6] Later, recurrences may be inaccessible for this study or may be less reliable if hormonal or radiation therapy has been used.[39] Inquiry is made about bone pain; appropriate x-rays and bone scan are obtained to rule out metastatic disease.

Transfusions are not regularly used, but cross-matched packed red cells should be ready. When the diagnosis of carcinoma is strongly suspected on physical and/or mammographic examination, and especially if proved by outpatient needle biopsy, autotransfusion is provided. As a part of the preadmission preparations, a unit of the patient's blood is drawn 3 or 4 days before the scheduled operation and reserved in the blood bank for her. Her own blood volume is restored in that time and

her marrow stimulated by the prior depletion. This blood is reinfused early in the operation, and is usually the only blood required. Concerns about hepatitis and incompatibility are thus reduced.

Coagulation defects, diabetes, cardiac, and pulmonary problems are identified and corrected. Electrolyte abnormalities (especially potassium depletion due to diuretics) are restored to normal. Intercurrent infections (skin, dental) are treated prior to the operation.

Intraoperative Complications

Incision and Flaps. The incision should not cross into the hollow of the axilla, as "bow-string" contracture may result. Skin flaps should be gradually tapered. Construction of flaps which are thicker at the edges than toward the base causes devascularization of the distal flap. "Button-holes" may be formed in such thinned-out areas; the defect is relatively unimportant, but the devascularization of the surrounding tissue is serious.

Skin hooks should elevate the free edge of the flap vertically, so that no eversion of the flap occurs at the dissecting angle. The surgeon can check the thickness of the flap by palpating it through a moist sponge. Sharp instruments are not used, except for fine skin hooks which make the early stages of flap formation easier. Elevation of the periosteum leads to bleeding which is difficult to control; it may also lead to new bone formation which may mimic a recurrence.

Pneumothorax. The cause of pneumothorax is penetration of the chest wall while securing the perforating vessels. This is best avoided by having the first assistant positioned opposite the operator where he can visualize and grasp the perforating vessels, preferably before they are divided. Straight hemostats are placed flat (horizontally) on the chest wall to avoid perforation. If these vessels escape or the hemostats twist, the vessels may retract and continued bleeding result.

Nerve Injuries. The intercostobrachial nerve is divided; this accounts for postoperative numbness of the axilla and opposing inner surface of the arm.

The thoracodorsal nerve is usually resected. Dissecting this nerve out of the axillary tissues impedes the axillary dissection, particularly when the nodes are involved.[15]

The long thoracic nerve must be spared or winged scapula deformity will result. This nerve is identified by dissecting the axillary tissue off the serratus fascia. The nerve is exposed parallel to its course. Gentle traction affords good exposure and the nerve is readily protected.

Vascular Injury. Injury to the axillary vein is the only major vascular injury encountered; the axillary artery is above the vein and beyond the boundaries of dissection. The vein is identified early in the axillary dissection beneath the deep layer of clavipectoral fascia (costocoracoid ligament) and below the cords of the brachial plexus (which can be palpated through the axillary tissue). Sharp dissection carried parallel to the course of the vein provides the safest approach. Each branch of the axillary vein is individually clamped in continuity, di-

vided, and ligated as encountered. Short stumps of the veins are left and each branch is ligated at its junction with the axillary vein. The axillary vein is pulled downward by the weight of the specimen; this tethering is most pronounced at each branch and distorts the vein, increasing the chance of injury. The axillary vein is not retracted. If it is lacerated, control by compression on either side of the defect permits visibility for suture.

CLOSURE OF THE WOUND. Tension is lethal to the wide thin flaps which are dependent on a random blood supply. The flaps can often be pulled together, but if tension is present, they will become pale or cyanotic. If such discoloration is present at the conclusion of the operation, sufficient sutures should be removed to relieve the tension. Even a small skin graft will prevent the loss of a considerable area of the flap. Skin grafts are sutured to the flap to prevent their shifting or rolling up. It is more important to get the patient moving than to limit arm and shoulder motion in an attempt to improve skin graft survival; the skin graft takes regularly.

Postoperative Complications

The overall mortality of radical breast surgery is about 0.4 per cent and wound complications have been reported as high as 39 per cent. Pulmonary and cardiac complications and venous thrombosis are about as frequent as in other similar major surgery.[42]

Flap necrosis of minor degree occurs frequently and is due to devascularization or trauma to the flaps or to tension. Local debridement usually suffices and the defect spontaneously epithelializes. A large necrotic area (greater than 3 cm) is best treated by excision and preparation of the area for closure by skin graft.

Hemorrhage in the immediate postoperative period is recognized by swelling and elevation of the flaps, large volumes of blood recovered from the suction catheters, and systemic signs of blood loss. Compression of the wound, aspiration of catheters, and replacement of blood are employed initially. If not successful, the wound must be reopened, clot evacuated, and bleeding source ligated. Even careful search will not always disclose the source, but the possibility of continued bleeding must be eliminated.

Fluid accumulation is avoided by fixation of flaps and suction catheter drainage. These steps minimize the dead space and reduce fluid accumulation. The catheters may be ineffective if they become plugged by clot or if the suction applied (e.g., wall or mechanical suction) is so great as to completely collapse the collecting cylinder. Occasionally, when fluid accumulation is evident and the catheters are not working, aspiration alone or aspiration followed by injection of a few ml of air into the catheters will dislodge clot and drainage will start. Any manipulation of the catheters must be done under sterile conditions.

Infection is usually related to fluid collection, flap necrosis, or wound separation. If fluid accumulates, it should be aspirated by syringe and needle. Aspiration directly through the flap provides a site of entry

for organisms. Antibiotics are not used routinely postoperatively. If repeated aspirations are performed or if healing is delayed by separation or flap necrosis, systemic antibiotics are given.

Drains predispose to infection. The defect from which the drain exits is also a potential entry site for organisms. Ordinary wick or tube drains are sealed off early and do not remove the fluid as well as suction catheters. Prolonged fluid accumulations are more likely to become infected not only because of the duration of the exposure, but also due to progressive decrease in opsonin activity of the fluid proteins.[1] Suction catheters, while creating a perforation of the flap, perform the necessary function of eliminating the fluid collection.

Wound separation may result if sutures are removed too early. There is no hurry about getting the skin sutures out. Any sutures which are under tension or show any redness are removed as signs appear. Interrupted closure permits removal of offending sutures. This is not possible if a continuous closure is used.

Early arm edema is said to occur in about half of mastectomy patients. The majority of patients develop some degree of edema, often so slight that they are unaware of it. Subtle changes such as shinier skin, less wrinkling of skin, and less prominent veins than of opposite hand are observed; measurements do not always reveal these minor degrees of edema. Edema evident after weeks or months is reported to be about 10 per cent[18] and is similar to our experience. Radiation therapy, wound infection, persistent seroma leading to fibrosis, and hand infections increase the frequency and severity of arm edema.

Early return to normal activity is encouraged to avoid limitations of arm and shoulder motion. Exercises are started the day after surgery. Three exercises are emphasized: (a) external rotation with hand placed behind the neck; (b) internal rotation by reaching behind the back to touch the opposite scapula; and (c) abduction of the arm, assisted by walking fingers up wall. The services of a well-trained visitor (herself a postmastectomy patient) from the Reach to Recovery program of the American Cancer Society provides a dimension of encouragement and support which is invaluable.

While encouraged to resume all their usual activities, patients are cautioned to protect the hand and arm by wearing gloves for such activities as gardening or working with tools. The skin is kept from excessive drying by use of a lanolin-based lotion, especially in winter. Manicure injuries, as in pushing back the nail fold, are avoided. Any break in the skin which becomes red is reported promptly and antibiotic therapy begun. Injections are not to be given nor is blood drawn from the affected arm. After the wound is healed there is no reason that the blood pressure cannot be taken on that side.

There is a reduced incidence of arm edema after modified radical mastectomy when compared with the standard radical mastectomy. This may be due to better venous and lymphatic return from the arm, but it has been suggested that such improvement is due to less complete removal of axillary nodes.[18]

Local recurrence within the flap may be an operative complication due to leaving a thick flap with breast tissue or implantation of tumor during the procedure. Haagensen[15] states that the parasternal and axillary recurrences are probably not attributable to the operation. Clinical recurrences should be biopsied to rule out pseudorecurrence. Proof that a lesion is in fact a recurrence is necessary, particularly prior to the institution of palliative treatment. Elevation of periosteum with new bone formation, inclusion cysts, fibromas, and suture granulomas are conditions which have been erroneously diagnosed as recurrent carcinoma.

SUMMARY

Initial diagnostic errors are related to the presumption that symptoms or findings are due to benign causes. Physical examination augmented by mammographic study will disclose those benign-appearing lesions which may harbor a carcinoma. All palpable lesions and mammographically suspicious areas require that their identity be determined. The final resolution depends upon biopsy.

Missing the tumor also causes diagnostic error. An accurate biopsy or "sample" must be obtained for study. Negative results of sampling techniques should be followed by formal biopsy.

Failure to eliminate the primary disease by leaving tumor or breast tissue behind increases the incidence of recurrence. Total mastectomy reduces this risk. It is our view that removal of the axillary nodes also aids in obtaining cure, as well as providing prognostic information. Well intentioned attempts to obtain a better cosmetic appearance or to reconstruct the breast are secondary to the patient's chief need which is to achieve cure.

Complications of the surgical procedures are due to improper flap dissection, desiccation and trauma to tissues, incomplete hemostasis, and inadequate drainage. Attention to operative details and adherence to well established surgical principles will minimize complications.

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Vascular Surgery

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The vascular surgeon deals with an elderly patient population and with a progressive metabolic disease, atherosclerosis. In addition, the surgeon attempts to reestablish flow in narrowed conduits, the medium flowing has the propensity to clot under certain conditions, and the pump propelling the flowing medium is often not functioning ideally. Yet despite these adverse conditions the vascular surgeon does a creditable job of maintaining functional circulation. This is due to a strict selection of cases, meticulous attention to details, and excellent preoperative and postoperative care. This article will of necessity deal not only with the care of the immediate problems created by the specific complications, but will also emphasize the prevention of these complications.

General

Preoperative and postoperative care is important in every surgical patient but in the vascular surgical patient it must be emphatically underscored. Elderly patients with atherosclerosis cannot tolerate mild degrees of hypoxia or hypovolemia lest potentially lethal cardiac, cerebral, or renal complications ensue. A vascular graft with minimal flow due to a compromised outflow tract may thrombose with a further reduction of flow due to cardiac failure.

Moreover, since the vascular tree is relatively rigid due to the atherosclerotic process, the difference between hypovolemia with hypotension and hypervolemia with congestive heart failure is often surprisingly little.

Careful monitoring in a modern postoperative unit will help prevent some of these situations. Intra-arterial pressures, blood gases, cardiac outputs and central venous or pulmonary pressures need not be used in every patient, but should be used in any questionable circumstances. Although the surgeon can do little to halt the relentless progression of atherosclerosis and its consequences, these consequences should be minimized during the operative period (Table 1).

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Table 1. *Survival of Patients with Multiple Risk Factors: Emergency Plus Elective Aneurysmectomy**

RISK FACTOR	30-DAY	3-YEAR	5-YEAR
Normal EKG, no angina	48/52(92)	26/40(65)	19/35(54)
Abnormal EKG, angina	19/24(79)	8/22(36)†	2/18(11)†
Abnormal EKG, angina, myocardial infarction	14/17(82)	6/16(38)†	2/14(14)†
Abnormal EKG, angina, cardiomegaly	6/7(86)	1/7(14)†	0/6(0)†
Abnormal EKG, angina, distal occlusive disease	9/12(75)	3/12(25)†	1/11(9)†
Abnormal EKG, angina, cardiomegaly, myocardial infarction	4/5(80)†	0/5(0)†	0/5(0)†

*Values are expressed as number of survivors/total number of patients, with percentages in parentheses.

†$P < .05$.

In patients over 70 years old undergoing aneurysmectomy (elective plus emergency cases), the 30-day mortality is acceptable regardless of the risk factors, but the long-term survival is poor due to the progress of the atherosclerotic process. (Adapted from Baker, W. H., and Munns, J. R.: Aneurysmectomy in the aged. Arch. Surg., *110*:516, May 1975).

AORTIC SURGERY

Reconstructive procedures upon the abdominal aorta are done for repair of aneurysms or to increase the flow of blood to the legs. Usually a graft of Dacron is placed between the infrarenal aorta and the end of either the aneurysmal disease or the occlusive disease. An endarterectomy may be combined with Dacron grafting or, less frequently, performed alternatively. Although each operation as well as the manifestations of the atheromatous disease processes are different, many of the complications are common to all and thus they will be discussed together.

Intraoperative Hemorrhage

Aortic hemorrhage usually occurs during the dissection of an aneurysm and may be the indication for the operation. Proximal control of the abdominal aorta may be obtained in one of several ways. Compression on the anterior surface of the aorta with either a sponge stick or a specially designed clamp temporarily controls the hemorrhage until the infrarenal aorta is controlled (Fig. 1). A Foley catheter inflated in the lumen of the aorta is an effective method of control if the lumen is smooth. A large vascular clamp can be blindly placed through the lesser sac to control the immediate subdiaphragmatic aorta. And finally the thoracic aorta can be controlled through a separate thoracic incision or through a diaphragmatic incision. These techniques are all effective in controlling hemorrhage and the surgeon should choose the method which he can perform the most expeditiously.

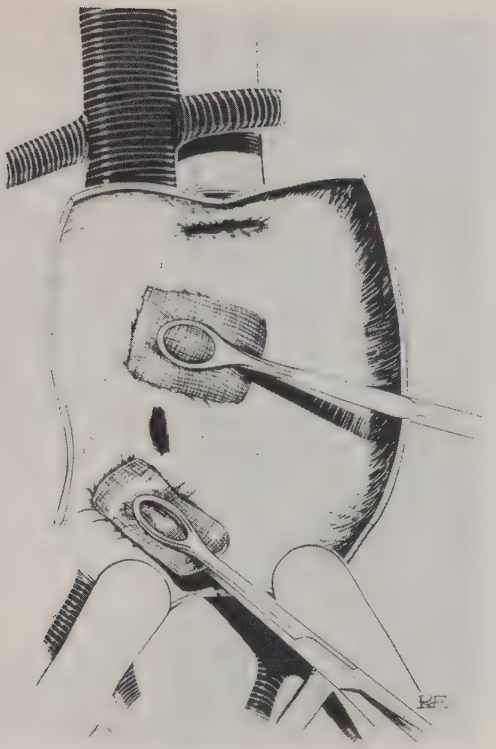

Figure 1. Sponge stick compression is an effective and quick method of either arterial or venous control. This figure depicts venous control of an aortocaval fistula secondary to a ruptured aneurysm. The fistula was then closed from within the aorta. (Reproduced from Baker, W. H., Sharzer, L. A., and Ehrenhaft, J. L.: Aortocaval fistula as a complication of abdominal aortic aneurysm. Surgery, 72:936, 1972. Used with permission.)

Venous bleeding most often comes from tributaries which are close to the inferior vena cava. The left renal vein should be identified anterior to the aorta and caudad to the superior mesenteric artery. If this vein is not seen in the usual anterior position, it can be in a retroaortic position, drain into a retroaortic lumbar renal vein plexus, or drain into a left-sided vena cava.[4] The left renal vein can be sacrificed to control hemorrhage if the collateral veins (adrenal or gonadal) are left intact. Lumbar bleeding behind the vena cava or the aorta can be life-threatening and for this reason complete aneurysmectomy has been abandoned in favor of leaving the posterior wall of the aorta in place and suture ligating the lumbar arteries from within the aorta. The surgeon should remember that venous bleeding occurs at low pressures and thus can usually be tamponaded temporarily until the vein is either ligated or repaired. If the major pelvic veins or the inferior vena cava require venorrhaphy, the repair must be technically perfect so that the suture line is not a source of a pulmonary embolus.

Intraoperative autotransfusion may be life-saving in cases of massive hemorrhage. It should be emphasized that autotransfusion is not

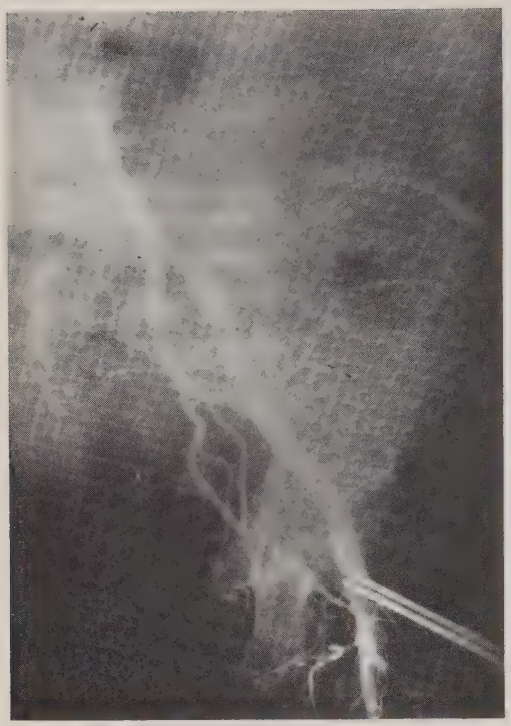

Figure 2. This operative arteriogram done after a femoral thrombectomy reveals a stenotic proximal external iliac artery stenosis. This could not be retrieved with a Fogarty catheter and a femoral-femoral bypass was then successfully performed.

without complications. Thrombocytopenia, hypofibrinogenemia, and hemolysis are the major complications, as well as air and possibly fat embolism. Newer modifications of the technique have minimized these problems.[29]

Intraoperative Thrombosis and Embolism

Distal thrombus may doom an otherwise satisfactory vascular reconstruction. This complication occurs when either atheromatous debris or intraluminal thrombus embolizes distally, or when prolonged vascular stasis encourages distal thrombosis. The former is commoner in aneurysm surgery and is a technical error which can be avoided by the gentle handling of the artery. Distal thrombosis should be prevented by the use of heparin. Intravenous heparin is distributed throughout the body and effectively gives protection against clotting (although excessive bleeding may be due to heparin overdose). Most surgeons empirically give 5000 U.S.P. units and supplement this with smaller doses if the period of vascular stasis is going to be longer than 30 to 45 minutes. The activated clotting time can be used to regulate this dosing schedule as well as postreconstruction administration of protamine sulfate. Locally injected heparinized saline is diluted by the flow from the first collateral artery and, whereas it is effective immediately distal to the clamp, it may not be effective distally in the leg. Finally when the clamps are released, flow is directed into the artery of least importance

(deep femoral artery as opposed to a patent superficial femoral artery) so that the effect of any emboli from within the graft will be clinically minimized.

If the surgeon suspects an arterial thrombus in the operating room because of the color of the feet, the absence of distal pulses, or abnormal pressures or flows, then it is a simple matter to either pass a Fogarty catheter distally or to obtain an arteriogram[25] to settle the diagnostic dilemma and initiate the appropriate therapy (Fig. 2).

Declamping Hypotension

When the vascular clamps are released after a Dacron graft is sutured into place, there may be a sudden drop in arterial pressure. This is especially true if the volume replacement during the first part of the operation has not been adequate. Release of the clamp may lead to bleeding through the interstices of the graft and may siphon off volume into the newly perfused vasodilated extremity. This complication can be minimized if the surgeon alerts the anesthetist and then slowly, notch by notch, releases the clamp.[17] If there is a sudden drop in pressure, the clamp can be reapplied until volume is restored and then released again. It is finally removed only when the patient can tolerate the physiologic changes.

Despite pressure changes, the surgeon cannot allow a stagnant column of blood to remain in the graft for long periods of time. Therefore the clamp must be released for a heartbeat or two every minute, lest clotting ensue.

In situations involving prolonged intraoperative ischemia there may be a build-up of lactic acid from the anaerobic metabolism of the extremity. A sudden flushing of this acidotic limb blood into the systemic circulation is said to contribute to cardiovascular instability.[26] Vasopressor factors have also been described.[6] However, the predominance of evidence minimizes these etiologies of declamping shock and emphasizes adequate volume replacement.[24]

Ischemic Colitis

Varying degrees of ischemia of the colon result in partial or full-thickness injury of the colon. Relatively mild ischemia produces submucosal hemorrhage with subsequent loss of epithelial integrity and ulcer formation, which results in a bloody diarrhea. Proctoscopically the rectum (blood supply via the internal iliacs) may be spared. The involved segments often have a friable mucosa with ulcerations, but the inflamed mucosa is said to lack the red hot intensity of idopathic ulcerative colitis.[2] "Thumb printing" may be seen on a barium enema examination and colonic dilation occurs relatively late.[19]

The anatomic area of the colon involved depends upon which major artery has been interrupted and upon the adequacy of the collateral circulation. During aneurysmectomy the inferior mesenteric is routinely sacrificed and thus the descending and sigmoid colon are at risk. The collateral circulation normally comes from the superior mesenteric artery via the middle colic artery, and the internal iliac arteries via the

hemorrhoidal arteries. If a prior colonic resection has been performed and the normal collaterals interrupted, colonic ischemia is more likely to occur.

A variety of surgical techniques has been described to prevent this complication. When the aorta is dissected at the base of the inferior mesenteric artery, the plane of dissection must be immediately adjacent to the aorta to prevent the inadvertent sacrifice of any inferior mesenteric collaterals. If the dissection is difficult, then the inferior mesenteric artery can be controlled from within the aorta at the time of aortotomy. A proximal end-to-side anastomosis keeps all of the collaterals intact. A replacement graft should be situated so that flow is maintained to the internal iliac arteries by anastomosing the distal end of a replacement aortic graft proximal to the internal iliac artery. In aortofemoral bypass grafting with a distal end-to-side anastomosis, the internal iliac artery will be fed via retrograde external iliac flow.

Recently the Doppler has been used in the operating room to detect flow in the pericolic arterial arcades.[36] Absence of flow with the inferior mesenteric clamped is an indication for reattachment of the inferior mesenteric artery. At the time of aortotomy a button of aorta including the inferior mesenteric artery is excised and reattached to an appropriate place on the graft.

Treatment of established ischemic colitis depends upon the degree of ischemia. Bloody diarrhea with no abdominal symptoms may be treated nonoperatively with the knowledge that a few patients will develop colonic strictures. Laparotomy with colonic resection is indicated by the usual clinical signs of abdominal pain, tenderness, fever, and ileus. Laparotomy should not be deferred until generalized peritonitis develops.

GRAFT OCCLUSIONS

Vascular reconstructions, regardless of their anatomic location, occlude for three basic reasons: poor inflow, an inadequate graft or endarterectomy site, and poor outflow. These principles apply to both acute and chronic occlusions. Despite the simplicity of these basics, it is often difficult to ferret out the exact etiology of each failure. This is especially true for the surgeon who thought that he had performed a technically perfect primary operation. Thus every time a graft fails the following ritualistic thought process is carried out.

Poor Inflow

The preoperative angiograms must be reviewed to look for lesions that were considered not important in the original evaluation. It is rare that a stenotic aortic atheroma is missed proximal to the insertion of an aortofemoral graft, particularly if the graft is sutured to the aorta in the immediate subrenal position. Inadequate inflow is more apt to be the reason for closure of a femoral popliteal graft. The grafts are more often placed for threatened limb loss in elderly patients with more widespread

atherosclerosis. Since they are elderly and have a variety of manifestations of vascular disease, laparotomy is avoided despite the presence of arteriographic aortoiliac disease if the femoral pulse is deemed satisfactory and pressure measurements indicate adequate inflow. However, these lesions, especially if they are combined with some decreased cardiac output, may be enough to doom the more distal graft.

Inadequate Graft

If inflow is adequate and the run-off is good, then perhaps the reason for graft failure is the graft itself or an inadequate vascular anastomosis. Operative arteriograms[25] are extensively used in this clinic to try to avoid technical errors, while others use pressure gradients and reduced arterial flows.[31] These latter methods will detect gross differences whereas the arteriogram will detect lesser degrees of an anastomotic narrowing.

Acute aortoiliofemoral reconstructive failures are rarely caused by the Dacron graft. The graft can twist as it exits along the iliac vessels; however, newer graft models have incorporated a stripe along the graft to lessen the chance of this complication. These grafts are sutured to relatively large arteries so that technically acceptable anastomoses are readily achieved. The large caliber of the graft limbs provides for excellent flow. Late closure of Dacron grafts can result from the development of thick "pseudo intima" or atherosclerosis. In my experience this usually occurs close to or at the suture line (Fig. 3).

Reoperations in the abdomen for aortofemoral graft failures require a surgeon skilled in dissecting in a scarred retroperitoneum. An operation which provides excellent inflow with less energy expenditure and less time consumption is a femorofemoral bypass graft from the patent

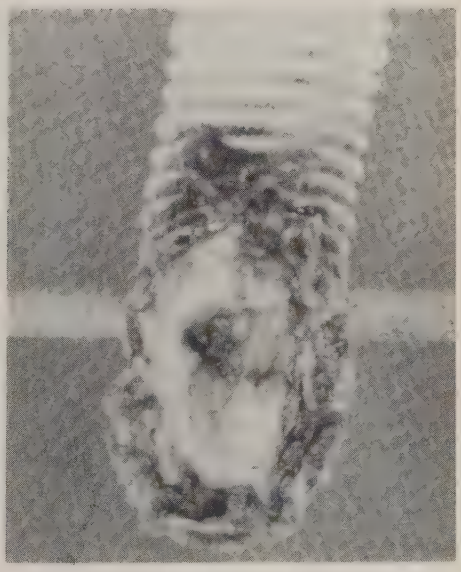

Figure 3. This photograph of an occluded graft demonstrates an overgrowth of atheromatous intima across the femoral anastomosis. A femoral-femoral graft restored the pulses to normal.

limb of the aortofemoral graft to a point distal to the previous graft insertion.[5] Not only is the operation simplified but the long-term results appear to be equally satisfactory.

Femoral popliteal occlusions are more often due to graft failure. Autogenous saphenous vein is the preferred arterial substitute. This is especially true if the knee joint is crossed and run-off is poor. Saphenous vein of less than 5 mm in diameter may lead to an increased failure rate although success with 4 mm veins is reported.[18] Alternatives to the saphenous vein include the cephalic vein, bovine heterograft, Dacron, and polytetrafluoroethylene. The arm must be prepped and draped ahead of time if the arm is to be used as the source of the vein. In addition, the arm vein must be protected from an enthusiastic horde of laboratory technicians, house officers, and anesthesiologists. The bovine heterograft is mentioned only to caution the unwary surgeon that reports[9] of a high incidence of graft failure and of late aneurysm formation have led to a recent condemnation of its use. Expanded polytetrafluoroethylene[15] is currently enjoying a wave of popularity, but again, no long-term results are available. Knitted Dacron grafts have been used with fair success above the knee but there is no enthusiasm for their use in the distal leg. Newer internal and external velour coverings of these grafts may or may not alter these opinions. Dardik et al.[10] report success using umbilical veins.

It is my current preference to use (1) saphenous vein, (2) composite grafts of veins from each leg, (3) a composite graft of polytetrafluoroethylene and saphenous vein if not enough vein is available, using the vein distally and the polytetrafluoroethylene proximally where motion is limited, (4) Dacron or polytetrafluoroethylene if the graft is above the knee, and (5) polytetrafluoroethylene for distal bypass. If I know that no saphenous vein is available preoperatively, then the arm is prepped so that the cephalic vein can be harvested. From a practical point of view, this does not happen very often but is a cogent argument for the use of preoperative venograms.

At the time of reoperation when a thrombectomy has been performed and an operative arteriogram reveals a technically satisfactory job has been done, the surgeon must obviously question his choice of a graft. "Should I have used vein from the contralateral leg or arm instead of the Dacron that was used?" "Should I have discarded the small vein graft in favor of a larger synthetic graft?" If the surgeon answers in the negative he implies that a constitutional factor incited clotting or else thrombus was retained from the time of the primary operation.

Inadequate Outflow

Whereas an acute graft failure following a properly indicated operation is most often due to some technical error, late graft occlusion on the other hand is most often due to poor run-off secondary to the progression of distal atherosclerosis, which relentlessly compromises the outflow tract.

Aortofemoral grafts should circumvent as much atherosclerosis as possible by being placed distally in the common femoral artery, or onto

the deep femoral artery if the origin of this vessel is involved with an atherosclerotic lesion. An alternative is to perform an endarterectomy at the time of graft placement. To ignore this principle may not lead to an increase in acute graft occlusions but will lead to an increased incidence of late graft failure.[21]

Likewise, in choosing a distal site for a graft bypassing a superficial femoral artery occlusion, the surgeon should bypass as much of the atherosclerotic process as possible. That is, go to the distal popliteal artery if there is a stenosis in the proximal popliteal artery, go to the posterior tibial artery (peroneal artery) if there is a stenosis in the tibial peroneal trunk, etc. Long-term results of these more distal grafts are equal or superior to the more proximally placed grafts, and the immediate failure rate should be less if the run-off distal to the graft does not contain a significant stenosis.[28] The run-off may be increased by making multiple distal anastomosis to more than one tibial (peroneal) artery.[11] There is another opinion which states that this distal bypassing is unnecessary and that a graft placed into a popliteal artery with poor run-off is all that is required to save a leg.[27]

When facing a patient with an occluded graft, good inflow, an apparently satisfactory conduit, and a high quality operative arteriogram that verifies a good technical result, the surgeon should review his choice of a distal anastomosis. If the deep femoral run-off is not satisfactory, then will the addition of a femoropopliteal bypass keep the graft open? If the popliteal run-off is unsatisfactory, then will the addition of tibial run-off keep the graft open? If the anterior tibial run-off is unsatisfactory, will an additional limb of the graft to the posterior tibial artery keep the graft open? None of these difficult questions have ready answers. These questions however are answerable in the operating room, and, if the primary procedure was properly indicated, a secondary procedure at the time of acute occlusion is likewise properly indicated. However, if the primary indication no longer exists (i.e., a foot ulceration healed, then the graft occluded), then there is no indication for the secondary procedure.

Finally, a chronic graft infection cannot be ruled out in patients with a late graft occlusion until the patient is explored. The surgeon must have an alternative surgical plan that includes possible graft excision and extra-anatomic bypass.

Graft Infection

An infected vascular graft is a complication with disastrous potential. These patients may present with an occluded graft, a false aneurysm, acute suture line bleeding, graft-enteric or urinary tract fistulae, or septic emboli. They may or may not be febrile or systemically ill.

Characteristically the graft is not incorporated into the fibrous sheath that surrounds the graft but instead this space contains a thin watery to frankly purulent fluid. Since preoperative antibiotics are liberally used by vascular surgeons, the Gram's stain obtained at exploration as well as the culture may be negative for organisms. These negative studies should not deter the surgeon from his clinical diagnosis. Treat-

ment of these patients in general requires exploration and excision of the infected graft.

If the patient has an aortoiliac graft, then this graft can be totally excised, the abdomen closed with a unit closure, and an axillofemoral bypass constructed through a clean field. A monofilament suture is used to close the infrarenal aorta, inserting a proximal row of three or four mattress sutures and a more distal row of continuous suture over the end of the aorta. If the graft has originated off the anterior aspect of the aorta, the aorta can be closed leaving a compromised lumen, to allow blood flow caudally. A patch angioplasty to widen the lumen is not advisable in the face of infection.

In the patient with an infected aortofemoral graft, the treatment possibilities are all compromises. If the surgeon excises the graft and the patient has two viable extremities, then he should terminate the procedure and accept symptomatic but viable legs. But if the legs are not viable, he is left with three poor choices: amputation at the lowest viable level; regrafting in the same infected area with synthetic material; or extra-anatomic bypass either in the same infected groins or distally in the leg with the graft traveling extremely close to the infected groins. These poor choices are difficult to judge. If the surgeon opts for amputation, he may be surprised that the level of viability is quite proximal and may involve the trunk and viscera. This is a terminal situation. On the other hand, the other two choices invite an infected graft. Although there are occasional reports of success using any of these methods, the morbidity and mortality of these operations are high.[34]

A third treatment choice is excision of the graft with endarterectomy of the patient's atherosclerotic arteries and drainage of the infected areas. An arteriotomy in an infected area seems heretical, although this technique is currently being successfully practiced.[37] Regardless of the surgical technique used, the importance of proper, prolonged antibiotic coverage cannot be overemphasized.

If only one limb of an aortofemoral bifurcation graft is clinically infected, does the entire graft have to be removed? In infection early after aortofemoral grafting, successes have been reported with unilateral graft resection,[34] but in late infections the septic process usually involves the entire graft. If neither the suture lines nor the pseudointima are infected, some authors[8, 34] recommend debridement of the perigraft tissue and irrigation with appropriate antibiotics. In my limited experience these patients will eventually re-present with an infected graft.

If the surgeon encounters a graft duodenal (jejunal) fistula, should he excise the entire graft or can he merely repair or replace the graft, as the duodenum may not harbor a plethora of virulent organisms. The literature is not clear on this issue.[12, 34] In my opinion, the decision rests upon whether or not the patient has an aortoiliac graft which lends itself to exicision and extra-anatomic bypass or an aortofemoral graft which may be excised but cannot be adequately bypassed through clean tissue planes. The same principles apply to the rarer aortoureteric and aortocolonic (appendiceal) fistulas.

CEREBROVASCULAR SURGERY

Carotid endarterectomy is performed almost to the exclusion of other cerebrovascular procedures. The dreaded complication of this operation is fixed neurologic deficit. In recent years the decreasing complication rate has approached 1 per cent,[13, 35] increasing the popularity of the operation and broadening the indications to include asymptomatic, stroke-prone patients. This section will deal with the operative indications, the treatment of complications, and finally review the details that are important in keeping the complication rate at a minimum.

Carotid endarterectomy is designed to prevent permanent neurologic deficit by removing an embolic source or maintaining normal cerebral flow. It is not designed to reverse established neurologic deficit, and in fact may worsen a patient with an acute stroke.[38] In an acutely stroked patient with an occluded internal carotid artery, the reestablishment of arterial pressure may convert a relatively bland ischemic infarct into an expanding hemorrhagic infarct with dire clinical results. Thus operation in a patient with an acute, fixed, neurologic deficit is contraindicated.

Carrying this argument a step further, a patient with a new, fixed, neurologic deficit after a carotid endarterectomy should not be reexplored. However, this conservative policy is not followed in all institutions. Others are more aggressive, stating that the surgeon cannot be sure that the neurologic deficit is fixed and that with the restoration of flow, neurologic function may return.[23] The data to support either position are not conclusive, for fortunately the number of postoperative strokes is small and treatment is based upon isolated case reports and not on well designed clinical studies.

The patient with new transient ischemic attacks postoperatively presents a different problem. In all likelihood thrombus either on the suture line or an imperfect end point is embolizing. If thrombus is causing the symptoms, then anticoagulants will control the symptoms. But on the other hand, if the patient does not have a fixed neurologic deficit, then he is also an operative candidate.

It is my policy to treat postoperative neurologic complications as follows. The patient is quickly examined. A palpable neck and superficial temporal pulse indicate that the common and external carotid arteries are patent. The flow in the ipsilateral supraorbital artery is then assessed with a Doppler ultrasonic velocity detector to determine abnormal flow patterns suggesting internal carotid occlusion.[7] Once the patency of the carotid arteries has been established, the postoperative patient is treated like any patient with a new neurologic deficit and reoperation is not performed. If the operated internal carotid is found to be occluded by the Doppler examination and the patient has a known occlusion of the contralateral internal carotid artery, then the patient is immediately returned to the operating room in an attempt to reestablish flow. This aggressive approach is justified by the known poor prognosis in patients with severe strokes and bilateral internal carotid occlu-

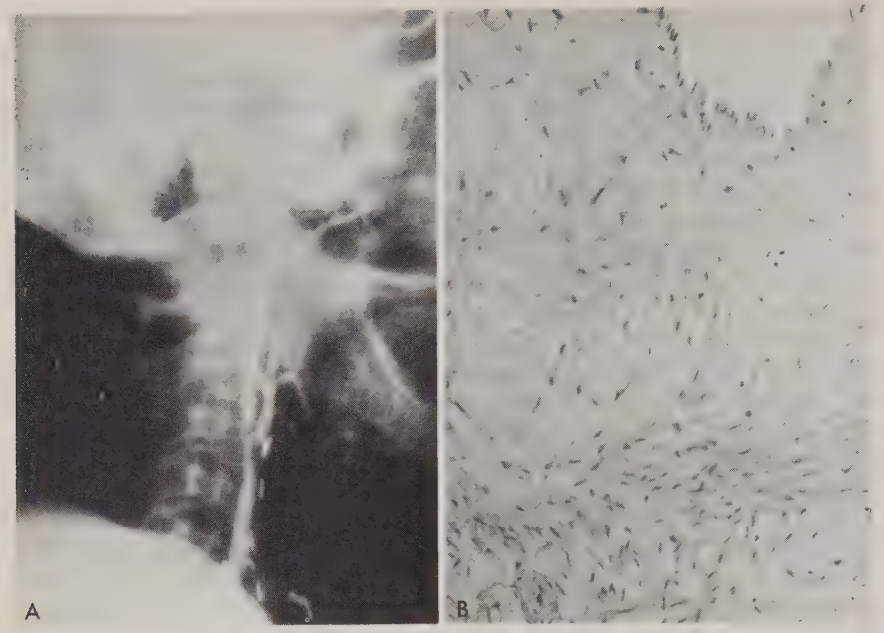

Figure 4. *A*, A symptomatic recurrent carotid bifurcation stenosis. *B*, Fourteen months after the original carotid endarterectomy which proved to be intimal fibroplasia. A limited endarterectomy with vein patch angioplasty corrected the lesion.

sions.[14] The postoperative patient with an ipsilateral occlusion and a contralateral patent internal carotid artery is treated nonoperatively as there is no evidence of the superiority of surgical treatment.

Patients with recurrent or new transient ischemic attacks in the early postoperative period should be studied angiographically and if a lesion is seen, this lesion should be treated appropriately. A poor distal end-point should be reoperated but a minimal clot on the suture line of an otherwise widely patent carotid bulb may be treated with anticoagulants.

Patients with the late recurrence of symptoms following carotid endarterectomy may have either an intimal fibroplasia of the endarterectomized segment, or the development of atherosclerosis, located either in the endarterectomized segment or distally in the internal carotid artery (Fig. 4). In either case the treatment is surgical and the procedure performed is usually some kind of endarterectomy with a patch angioplasty closure of the carotid bifurcation. False aneurysm following endarterectomy occurs rarely (Fig. 5).

My conservative reoperative principles are based upon my prejudice that most neurologic complications are probably embolic in nature with relatively few being due to immediate closure of the endarterectomized segment. The surgeon must constantly remind himself to "dissect the patient away from the artery" and to minimally manipulate the carotid bulb. The carotid bifurcation should be assumed to be full of friable thrombus in each and every patient. The common, external, and distal internal carotid arteries are controlled in areas that are palpably soft but

Figure 5. Two months after a carotid endarterectomy this patient returned with a pulsatile neck mass. At operation the suture was found to be broken. There was no evidence of infection. The aneurysm was replaced with a vein graft.

the carotid bifurcation itself is not dissected until the clamps have been applied to minimize the chance of embolization. After the endarterectomy has been completed, the distal end-point is inspected and if there is any question as to whether or not it is adherent to the media, it is surgically attached with fine suture material. The closure is completed without narrowing the artery. Although some surgeons routinely close the artery using a vein patch or Dacron patch angioplasty, I have not found this to be necessary. An operative arteriogram will verify a good result or pinpoint technical imperfection.[30]

Cerebral protection during the period of carotid clamping remains a controversial subject. All surgeons agree that the patient should have his blood pressure maintained in the high normal range and that oxygenation should be optimal. (This should be continued in the postoperative period.[20]) General anesthesia depresses cerebral metabolism while local anesthesia allows the surgeon to continually assess the patient's cerebral function. There appears to be no advantage of hypocarbia or hypercarbia.[1] A temporary indwelling shunt during the period of carotid clamping maintains cerebral flow and is a stent over which the carotid can be closed.[35] Others do not use the shunt as technically the operation is easier without its presence, the shunt itself can cause an

Table 2. *A listing of the Miscellaneous Complications Occurring in 304 Consecutive Carotid Endarterectomies*

Temporary nerve paresis	19
VII. 2	
X. 9	
XII. 8	
Bleeding, requiring exploration	4
Myocardial infarction	3
Electrolyte imbalance	2
Wound infection, psychosis, pneumonia, corneal abrasion, respiratory arrest, ruptured aneurysm	1 each

embolic stroke during its insertion,[33] and its superiority has not been proven. A third group of vascular surgeons selectively uses the indwelling shunt depending upon the internal carotid back pressure[16, 22] or the EEG.[33] Comparable results have been obtained by authors using all of the above methods.

Non-stroke complications include wound problems and paresis of the hypoglossal, vagus, and spinal accessory nerves and the mandibular branch of the facial nerve (Table 2). These temporary pareses are usually due to operative trauma to the nerve from surgical instruments including retractors and are preventable. Rarely, an anatomic variation, usually involving the premature branching of the recurrent laryngeal nerve from the vagus nerve, is encountered. While it is beyond the scope of this article to recount all of these possibilities, the surgeon performing cervical operations must be aware of these anatomic variations.

Operations other than carotid endarterectomy are infrequently performed for cerebrovascular symptoms. Intrathoracic operations are effective but because of improved survival rates of extra-anatomic bypass in the neck, carotid-carotid, subclavian-carotid, subclavian-subclavian, and axillary-axillary bypass procedures are increasingly used. When complications occur in these patients, the physician must integrate a variety of surgical principles into his treatment plan. His decision if or when to reoperate depends upon the neurologic status of the patient. The type of the reoperation will depend upon the etiology of the graft failure as discussed in detail elsewhere in this article.

EPILOGUE

Because of the constraints of space this article was not intended to be an encyclopedia of vascular complications but rather a compendium of common problems facing the practicing surgeon. Notable by their absence are the complications of balloon pumping, embolectomy, sympathectomy, amputation, thoracic outlet, renal and visceral reconstructions, arteriovenous fistula, and trauma. Nonetheless, many of the general principles enumerated in this article can be applied to other unnamed specific complications encountered in a busy vascular surgical practice.

The most important principle is a dedication to surgical excellence that will make an article on "Complications" a vestigial resource.

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Venous Disease

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Venous disease is common. In 1966, the Task Force on Thrombosis of the National Research Council concluded that venous thromboembolism was the leading cause of morbidity and mortality in the aging population of the nation.[26] Moreover, varicose veins are common in Western countries[6] and often require surgical management. Complications from venous disease or its treatment present challenging problems to the general and vascular surgeon.

VENOUS THROMBOEMBOLISM

Superficial Thrombophlebitis

Thrombophlebitis of a superficial vein is easy to diagnose by the presence of a firm, palpable, tender cord corresponding to the vein. Superficial phlebitis tends to be self-limited and commonly seen in patients with varicose veins. Occasionally, it may be troublesome because it is painful, and may cause undue worry to the patient because of fear of pulmonary embolism. When superficial thrombophlebitis occurs unassociated with varicose veins, the possibility of occult carcinoma should be considered.

Unlike thrombosis of the deep venous system, superficial thrombophlebitis seldom results in emboli to the pulmonary vasculature. On rare occasions, the thrombus may propagate in the superficial venous system to the femoral vein or the popliteal vein, and present with pulmonary embolism. The incidence of this complication, however, is very low and has been reported at 1 to 2 per cent.[30] To avoid this complica-

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Table 1. *Indications for Contrast Venography in Patients with Suspected Acute Deep Vein Thrombosis*

1. Equivocal noninvasive study in a patient with strong clinical suspicion of deep venous thrombosis.
2. A definitive proof of the presence of pulmonary embolism in a patient in whom there are no leg symptoms and a normal noninvasive test.
3. Prior to surgical interruption of the vena cava for pulmonary embolism.
4. Evaluation of recurrent venous thromboembolism in a patient who has had
   a. Previous vena cava surgery
   b. Previous documented evidence of acute deep vein thrombosis.

tion, the saphenous vein at the groin should be ligated when thrombosis propagates above the midthigh. No resection of thrombosed vein is necessary.

Acute Deep Vein Thrombosis

Unlike superficial thrombophlebitis, pulmonary embolism following acute deep vein thrombosis is common. Diagnosis of acute deep vein thrombosis may be difficult and the fallibility of clinical signs in establishing the diagnosis has been emphasized recently by several investigators.[4, 13] Diagnostic accuracy is improved by the use of noninvasive tests with the transcutaneous Doppler ultrasound technique,[96] the impedance plethysmograph,[94] and the phleborheograph.[12] These tests are used for screening and must be employed in conjunction with clinical judgment.

When the results of noninvasive testing are at variance with clinical signs and symptoms, contrast venography is the standard objective method to establish the diagnosis. Indications for contrast venography are listed in Table I.

Disadvantages of venography include the cost of radiographic equipment and patient discomfort. Complications appear to be limited to occasional local extravasation of contrast medium at the needle puncture site and, rarely, allergic reactions to contrast medium. Fatal allergic reaction is rare and is estimated to be similar to the incidence reported for intravenous pyelography (1 per 50,000).[25] The contrast medium is irritating to the intima of veins and must be flushed from the leg veins with heparin solution after completion of the procedure. Even with this precaution, postvenography thrombosis, pain, tenderness, or swelling on the day following the venogram has been observed by the authors as well as others.[11, 66, 72] Recently, Albrechtsson and Olsson[2] reported increased uptake of ^{125}I-fibrinogen in 20 of 61 patients with initially normal venograms. Four of these 20 patients (7 per cent) had evidence of thrombosis or thromboembolism on lung scan or repeat venogram.

^{125}I-fibrinogen scanning may also be used to detect calf vein thrombosis. The technique is best suited to studies of the epidemiology of venous thrombosis and evaluation of methods for its prevention. Following the administration of radioactive fibrinogen, an actively growing thrombus will incorporate the radioactive material, allowing detection

of the thrombus with a portable scanner. A major disadvantage of this procedure is its poor sensitivity in the detection of thrombus in the thigh, groin, or pelvis because of high background activity. Since thrombi may arise de novo in the profunda, pelvic, or internal iliac veins, as well as by extension from the popliteal and calf veins, a negative fibrinogen uptake is not an absolute indication that venous thrombosis is not present (Fig. 1). False negatives may also result if the thrombus is fully formed when the fibrinogen is administered, since the radioactive material will not be incorporated into this static thrombus. The ^{125}I-fibrinogen technique is very sensitive and false positives may occur with superficial thrombophlebitis or hematomas. Because of these problems, this technique is not optimal for use in the evaluation of the patient with suspected deep venous thrombosis. Its greatest utility has been in the detection of calf vein thrombosis following surgical procedures and determination of the efficacy of prophylactic measures such as low dose heparin, mechanical compression, and electrical stimulation of the muscles. A potential complication of this procedure is serum hepatitis secondary to fibrinogen. Kakkar[47] found no serum hepatitis result-

Figure 1. Venogram of a patient presenting with acute left iliofemoral vein thrombosis. Thrombus is seen in the left iliac vein (*arrows*). The femoral, popliteal, and calf veins are normal.

ing from 2000 examinations. The fibrinogen was prepared from a small pool of five donors who had donated five previous transfusions without evidence of hepatitis in the recipients in a 6-month observation period. Each batch of serum was screened for the presence of Australia antigen. Similar precautions in the preparation of ^{125}I-fibrinogen should make this a rare complication of this diagnostic technique.

Once the diagnosis of acute deep vein thrombosis is established, the treatment of choice is the administration of intravenous heparin, followed by a period of anticoagulation with oral agents. Thrombectomy is now seldom performed because of high recurrence rates. However, sudden massive venous thrombosis may present with symptoms simulating acute arterial insufficiency and then requires thrombectomy. This type of venous thrombosis, called the "ischemic form of acute venous thrombosis."[40] usually involves both the iliac and femoral veins. If untreated, it may progress to gangrene of the toes or foot. The ischemic form of venous thrombosis is not infrequently seen in patients with carcinomatosis.

The therapy of deep venous thrombosis is aimed at the reduction of acute symptoms and the prevention of the sequelae of pulmonary embolism and chronic venous insufficiency. Anticoagulation is efficient in attaining these goals but is not without morbidity and occasional mortality.

Since heparin cannot be absorbed from the gastrointestinal tract, it must be administered parenterally by intravenous bolus or by continuous intravenous infusion. Current studies favor continuous intravenous infusion, which is associated with a lower incidence of bleeding.[82] Control of the infusion rate with an electronically metered drip assures a constant infusion rate.

Complications associated with the use of anticoagulation in the treatment of deep venous thrombosis include hemorrhage,[82] rare allergic reactions,[89] thrombocytopenia,[73] arterial emboli,[77] and local skin necrosis with Coumadin derivatives.[68] Coumadin-induced intestinal obstruction due to hematoma in the bowel wall or mesentery is not an uncommon complication.[31] Major bleeding from either the gastrointestinal tract or the urinary tract must be further investigated for evidence of pathology in these two systems.

Surgical procedures and trauma increase the risk of significant bleeding from anticoagulation. Of 33 postoperative patients who received heparin in Lewis and Dale's series,[54] four (12 per cent) bled from the operative site. Limitations of the number of venous and arterial punctures and muscular injections will significantly reduce the number of bleeding episodes. The increased risk of bleeding with anticoagulation in the postoperative patient diminishes with time. Heparin is not contraindicated in this group of patients, but careful observation for bleeding is imperative. In addition, the type of heparin and the dose expressed in milligrams (mg) must be carefully inspected prior to administration. Because the strength of heparin varies with the origin (gut or lung), therapeutic dosage must be calculated in USP units, rather than mg, thus avoiding overdose and unnecessary bleeding. Heparin,

given in a dosage of 100 units per kg, is sufficient to institute full heparinization. Monitoring of the dose required to maintain anticoagulation can be achieved by daily measurement of partial thromboplastin time (pTT), whole blood clotting time, or the Lee-White test. The incidence of bleeding is better correlated with the mode of administration than the type or frequency of the monitoring test. However, in patients with hepatic or renal disease, frequent monitoring and smaller doses of heparin may be necessary to reduce the incidence of bleeding.

Thromboembolism during anticoagulation is a serious but infrequent complication, occurring in less than 5 per cent.[1] Heparin prevents extension of the thrombus, allowing lysis or fixation to the vessel wall. Despite resolution of this acute process, the risk of recurrent thrombosis and pulmonary embolism remains high immediately after hospital discharge, reaching a plateau at 3 years.[9] Because of the parabolic decrease in the risk of recurrent phlebitis and embolism during this period, Coon and Willis[9] recommended continuation of oral anticoagulants for 4 months following deep venous thrombosis to reduce the complications of recurrence.

Two thrombolytic agents, streptokinase and urokinase, have been used in the treatment of venous thrombosis and pulmonary embolism. Strict coagulation control by a hematologist is prerequisite for this form of therapy. Hemorrhage or allergic reaction are two common complications. Urokinase is less antigenic but very expensive and it is doubtful, therefore, that it will be used as a routine mode of therapy for deep venous thrombosis. The urokinase trial[90] found no evidence that mortality rates from pulmonary embolism were altered by treatment with these drugs.

Untreated or unrecognized deep venous thrombosis often progresses to chronic venous insufficiency as a result of damage to the venous valves. Postphlebitic syndrome is a disabling condition manifested by pain, edema, swelling, and skin changes from induration, pigmentation deposition, and painful ulceration.

Venous ulcer must be treated conservatively. At present, there is no evidence that any special local medication applied to the ulcer is of value. In fact, indiscreet use of various commercial products may sensitize the skin, causing eczema. Skin irritation and superimposed infection may lead to extension of the venous ulcer. The treatment of a venous ulcer is firm compression. An infected ulcer is treated by wet-to-dry dressing or surgical debridement. Skin grafting is seldom employed except when the ulcer area is large.

Prevention of Venous Thromboembolism

Prevention of complications of venous thrombosis begins with the identification of the high-risk patient and institution of appropriate prophylactic measures. An understanding of the epidemiology of venous thrombosis is important and information gathered by the ^{125}I-fibrinogen uptake test on the incidence of venous thrombosis is now available.

Patients over 60 years of age are prone to develop venous thrombosis in the postoperative period. Thrombosis occurs early and may even

Table 2. *Incidence of ^{125}I-Fibrinogen Documented Deep Vein Thrombosis**

SURGICAL PROCEDURE		INCIDENCE
General surgery	Over 60 years of age	60 per cent
	40 to 60 years of age	35 per cent
Orthopedic surgery		54 per cent
Urologic surgery		28 per cent
Gynecologic surgery		18 per cent

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begin intraoperatively at the calf level.[67] The incidence of thrombosis varies according to the type of operation performed (Table 2). Once the thrombus is formed in the calf veins, there is a 20 per cent chance of further propagation into the popliteal or femoral vein.[67] Many patients have spontaneous resolution of calf vein thrombosis. Although the usual risk factors, such as age, obesity, and smoking, may help physicians to decide when prophylactic measures are needed prior to a surgical procedure, a hematologic test would be ideal to identify the patient prone to thrombosis. Recent reports on measurement of serum antithrombin III level[20] and various hematologic tests[7, 8, 57, 87] indicate that these methods may help to identify the patient with an increased risk of thromboembolism.

Various prophylactic measures to prevent venous thrombosis are now available and the ^{125}I-fibrinogen technique has helped to clarify the value of these procedures (Table 3). The choice of these methods, however, may still be difficult. The decision must be based on the resources of the institution, the type of surgical procedure, the acceptance of the instrumentation by the patient, and the cost-effectiveness of the method.

Small dose heparin, as suggested by de Takats more than 2 decades ago,[15] has been shown to be effective in preventing postoperative deep vein thrombosis. Enthusiasm for the use of small dose heparin that prevails in Europe has not been equalled in this country.[10, 88] Lack of acceptance of small dose heparin as a routine prophylactic measure is explained by several factors. First, postoperative pulmonary embolism occurs infrequently. A report from Case Western Reserve University Hospital has noted an incidence of less than 0.1 per cent.[53] Of 54,183 adult patients discharged from the University Hospital of Cleveland during a 2-year period (1972-73), only 197 or 0.4 per cent were diagnosed as having pulmonary embolism, and of these only 32 (16 per cent) occurred in postoperative surgical patients.

Second, a positive fibrinogen uptake test may be a natural event following an operative procedure and calf vein thrombi detected by the ^{125}I-fibrinogen test may spontaneously dissolve[45] and are seldom of clinical significance.[18] Last, bleeding from small dose heparin, especially in orthopedic, gynecologic, or urologic procedures, may cause more mor-

Table 3. *Prevention of Postoperative Deep Venous Thrombosis**

METHOD	DVT DEPRESSION
Mechanical	
Elevation of legs on operating table	Not significant
Static compression of legs	Not significant
Static graduated compression of legs	47 per cent
Passive exercise of legs	77 per cent
Electrical stimulation of calf muscles	61 per cent
Intermittent compression of arms	60 per cent
Intermittent compression of legs	82 per cent
Intermittent compression of legs in cases of malignancy	89 per cent
Chemical	
Aspirin	Not significant
Dextran	Variable
Warfarin	Variable
Dipyridamole	Not significant
Dihydroergotamine	77 per cent
Subcutaneous heparin	80 per cent
Combinations	
Oxyphenbutazone and static compression	65 per cent
Intermittent compression ± subcutaneous heparin	†
Dihydroergotamine and subcutaneous heparin	77 per cent
Aspirin and dipyridamole	56 per cent

*Reprinted from Cotton, L. T.: Prevention of postoperative deep venous thrombosis. Br. Med. J., 4:1193, 1976. Used with permission.

†Not significantly different from intermittent compression alone.

bidity from bleeding than it prevents.[50] Even in patients treated with low dose heparin, the incidence of isotopic calf vein thrombosis in most reported series is substantial (Table 4). Only Nicolaides and his colleagues are able to produce a surprisingly low incidence of 0.8 per cent. Following orthopedic procedures, prophylaxis of venous thrombosis with dextran or warfarin appears to be superior to the use of low dose heparin because of a lower incidence of associated bleeding.[41, 81]

Other forms of prophylactic measures such as mechanical compression are cumbersome and need further study to ascertain whether reduced calf vein thrombosis indeed decreases the incidence of pulmonary embolism. Frequently, patients lose interest in the instrument, especially after the first or second day after operation, because of discomfort caused by the instrument. Furthermore, the effectiveness of physical measures has not been proved for patients at high risk of venous thromboembolism.[80]

Low dose heparin is less effective in prevention of venous thromboembolism in orthopedic,[81] gynecologic,[59] or urologic[42] procedures. In addition, alarming bleeding or hematoma formation may result from the administration of small dose heparin. Because of these reports, others have sought other pharmacologic means to prevent venous thromboembolism in these patients. The use of Dextran-70 has been reported to

Table 4. *Incidence of Isotopic Venous Thrombosis in Patients Treated with Low Dose Heparin Reported by Various Investigators*

	NO. PATIENTS TREATED	PER CENT ISOTOPIC THROMBOSIS
Williams[95]	27	15 per cent
Gordon-Smith, et al.[32]	52	13.5 per cent
Kakkar, et al.[48]	39	8 per cent
Nicolaides, et al.[67]	122	0.8 per cent
Gallus, et al.[27]	108	1.9 per cent
Lahnborg, et al.[51]	58	3.4 per cent
Ruckley, et al.[78]	125	12 per cent
Van Vroonhoven, et al.[92]	50	2 per cent
Sagar, et al.[79]	25	32 per cent
Gruber, et al.[35]	83	13.2 per cent
McCarthy, et al.[59]	64	10.9 per cent
Kakkar, et al.[49]	625	7.7 per cent
Pollock[70]	55	16.9 per cent

reduce pulmonary embolism but not the incidence of deep vein thrombosis.[5, 36] Several investigators have found warfarin to be highly effective in hip surgery.[46, 64, 76]

Obviously, all current suggested prophylactic measures are of value in the reduction of deep vein thrombosis but not necessarily in reduction of pulmonary embolism. It must be emphasized that an understanding of the natural history of calf vein thrombosis detected by the isotopic scan is important. Several investigators have stressed that a positive isotope scan, and hence the so-called calf vein thrombus, is seldom the cause of fatal embolism.[44, 71] Of 379 surgical patients studied, Doouss[18] has found 33 per cent with calf vein thrombosis but clinical follow-up indicated that the great majority of these patients suffered no ill effects. It would appear that prophylactic measures must be tailored to those patients who are at high risk rather than given as a routine regimen to all patients.

Noninvasive tests such as impedance plethysmography or Doppler ultrasound are now easily available and may allow close monitoring of the progression of the thrombus from the calf region to the popliteal level. If these tests become positive, a phlebogram should be done to further evaluate the condition and allow institution of proper treatment. This approach is probably better than giving anticoagulants to all patients.

Preventive measures should be administered to selected patients. Consideration should be given to that patient's likelihood of developing fatal pulmonary embolism if the preventive measure is withheld.

Pulmonary Embolism

If recent reports[63] are correct, approximately 15 per cent of deaths in acute general hospitals can be related to pulmonary embolism and this would increase the projection for the United States to the range of

126,000 cases per year. Diagnosis of pulmonary embolism may be difficult because of the ambiguity of presenting symptoms. Unless the clinician is aware of the varied presentation, the disease process may go unrecognized.

Since pulmonary embolism frequently occurs as a complication of extremity deep vein thrombosis, a careful examination of the lower extremities combined with noninvasive tests such as Doppler ultrasound and impedance plethysmography may localize the source of the embolus even when few symptoms exist. Even if noninvasive testing is normal, a phlebogram may be necessary to locate the nonocclusive type of thrombus which is most likely to give rise to pulmonary embolism.

An initial pulmonary embolus is rarely fatal (11 per cent). However, when massive pulmonary embolism occurs, death may result within an hour. Therefore, massive pulmonary embolism must be treated promptly with heparin, without delay for nonessential diagnostic work-up. If cardiopulmonary decompensation is apparent, the patient should have pulmonary angiography followed by pulmonary embolectomy. The classification of pulmonary embolic disease as suggested by Greenfield[33] should help surgical considerations of this fatal disease. Indications for open pulmonary embolectomy are those suggested by Sasahara,[83] which include systolic pressure less than 90 mm Hg, urine output less than 20 ml per hour, and PO_2 less than 60 mm Hg after 1 hour of nonoperative management. Because of high mortality associated with open embolectomy, a transvenous catheter embolectomy has been devised by Greenfield[33] and such a procedure offers an alternate approach to patients who are desperately ill. Preliminary use of this technique appears promising.

Fortunately, most initial pulmonary emboli are not fatal and anticoagulation is effective in preventing recurrent emboli. Indications for surgical intervention include failure of anticoagulation, a large nonocclusive thrombus in the iliac or femoral vein, recurrent chronic pulmonary emboli, septic emboli, and massive pulmonary embolism (Table 5). The preferred level of venous interruption is the inferior vena cava, below the renal veins.

Since Homans[44] suggested inferior vena cava ligation in 1934, many techniques for vena caval interruption have been introduced, including filters,[16] plication,[85] clip,[60] and transvenous methods of placement of umbrella[62] or filter.[34] Regardless of the method to be used, vena cava interruption must be preceded by a venogram to locate the source of the

Table 5. *Indications for Surgical Interruption of the Vena Cava*

When significant pulmonary embolization occurs
1. After adequate anticoagulation
2. When anticoagulants are contraindicated
3. Chronically on multiple occasions
4. Nonocclusive thrombosis of iliac or femoral vein

When anticoagulants given to prevent pulmonary embolization must be discontinued
When septic thrombophlebitis is uncontrolled
When pulmonary embolectomy is required

embolus. If the vena cava is totally occluded, retrograde venography via the brachial vein may be necessary. Judicious use of the venogram will allow the surgeon to be aware of the possibility of vena caval duplication or other anomalies, and also to assess the extent of the thrombi. If a loose tail of thrombus is seen in the upper vena cava, thrombectomy with vena caval clipping may be necessary (Fig. 2).

Vena cava interruption is associated with a mortality varying from 5 to 40 per cent.[84] Mortality rates reflect the cardiopulmonary status and underlying disease process of the patient population. Death rarely results as a direct consequence of the operative procedure. Hemorrhage is the most frequent operative complication, resulting from technical errors during the procedure. Extensive bleeding may result from avulsion of lumbar veins during caval dissection.

The major complication of all forms of vena cava interruption is the occurrence of venous stasis in the lower extremities. Frequency of postoperative stasis sequelae following cava interruption has varied in reported series from 10 to 50 per cent.[69] Several series have reported a

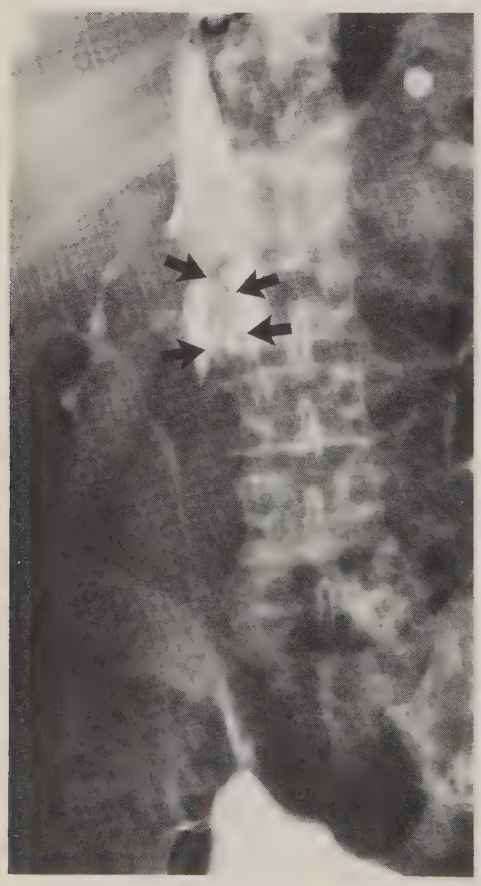

Figure 2. Abnormal venogram demonstrating a thrombus in the vena cava with loose nonocclusive clot (*arrows*) seen above the renal veins.

Figure 3. Thrombus developed in a patient who had vena cava clipping procedure. The patient had recurrent pulmonary embolus.

similar incidence in postoperative edema following vena caval ligations and partial interruption procedures.[69] The occurrence of postoperative edema is better correlated with the presence of preoperative edema and the extent of venous thrombosis than with the type of vena cava interruption performed. Although lower extremity edema is a common problem, permanent disabling venous stasis is rare (9 per cent).[69]

Anticoagulation in the postoperative period may decrease the morbidity from lower extremity edema by preventing extension of the venous thrombosis. Edema may be minimized or prevented by early institution of leg elevation and wrapping of the lower extremities in firm bandages.

The incidence of recurrent pulmonary embolism following cava interruption varies from 0 to 50 per cent in the literature and is dependent upon the patient population, length of follow-up, and technique used to diagnose recurrent embolization. Sources of recurrent emboli include the lower extremities via dilated collaterals, clots developed above the clip, and, rarely, the upper extremities and right heart (Fig. 3). When re-

current embolization occurs, a venogram and inferior venacavagram should be obtained to identify the source of emboli, venous anomalies, and dilated collateral pathways.

Prompted by reports of high mortality associated with caval interruption in cardiac patients, Mobin-Uddin[61] designed a transvenous method of caval interruption which could be performed under local anesthesia. The "umbrella filter" has now become an important form of caval interruption, especially in patients who are too ill for surgical intervention under general anesthesia. The use of the umbrella filter or steel filter,[33] however, has brought new problems. Complications of this form of interruption include filter dislodgement and migration, misplacement of the filter in a renal vein or iliac vein, peritoneal hematoma, and retroperitoneal perforation of the duodenum and ureter. Umbrella filter is not recommended in patients with septic emboli or in those with septicemia.

Filter migration has been decreased by an increase in the size of the umbrella filter from 23 to 28 mm. Complications resulting from malpositioning in the renal or iliac vein are decreased by combination of inferior cavography with filter insertion.[61] In order to prevent recurrent embolization following iliac ejection of the umbrella filter, a second umbrella may be positioned in the cava at the proper level. Anticoagulation should be discontinued prior to umbrella insertion in order to decrease the risk of retroperitoneal hemorrhage.

Surgical interruption of the vena cava is associated with a higher morbidity and mortality than anticoagulation therapy. It should therefore be employed only when specific indications exist. Vena cava interruption with a clip or ligature is preferred for patients who can tolerate a general anesthetic.

COMPLICATIONS OF THE SURGICAL TREATMENT OF VARICOSE VEINS

Varicose veins are the most common venous problem presenting to the surgeon. Untreated varicosities may present with complications like eczema or ulceration. Infrequently, external bleeding may cause a problem.[23] Current surgical management includes ligation of the greater saphenous vein with its tributaries at the saphenofemoral junction combined with saphenous vein stripping and ligation of incompetent perforators. Varicosities of the lesser saphenous system are treated by ligation at the level of the popliteal vein with stripping.

Complications of varicose vein surgery are few and usually minor. Natali[65] reported only 18 deaths in 87,000 patients. Likewise, the incidence of thromboembolic complications is extremely low, ranging from 0.4 to 0.7 per cent (Table 6). In 4080 patients operated on at the Mayo Clinic in a 10-year period, 16 episodes of nonfatal pulmonary embolism occurred.[56]

The most frequently encountered complications are wound-related: hematoma, wound infections, and skin necrosis. Less frequent compli-

Table 6. *Personal Results of Varicose Vein Operations**

	MORTALITY	VENOUS THROMBOSIS	PULMONARY EMBOLISM	INFECTIONS
Dale (557 patients, 1964–1974)	0	15 (2.6 per cent)	4 (0.7 per cent)	2 (0.4 per cent)
Cranley (2838 patients, 1952–1974)	0	–	3 (0.1 per cent)†	2 (0.07 per cent)
DeWeese (186 patients,‡ 1958–1968)	0	2 (1.1 per cent)	0	1 (0.5 per cent)

	Results	*278 patients re-examined*	*656 patients self-examined*
Myers	Excellent	44 per cent	48 per cent
10-year period	Good	41 per cent	38 per cent
	Fair	15 per cent	11 per cent
	Poor	0	2 per cent

*Reprinted from Dale, W. A., Cranley, J. J., DeWeese, J. A., and Myers, T. T.: Symposium: Management of varicose veins. Contemp. Surg., 6:86, 1975. Used with permission.

†Minor, nonfatal.

‡431 patients were evaluated.

cations include excessive blood loss, edema secondary to lymphatic disruption, saphenous or sural nerve damage, and major venous and arterial injuries. Prevention of wound complications is initiated in the preoperative period. Chronic venous ulcers which harbor infection with mixed organisms should be adequately treated prior to varicose vein surgery to prevent contamination of the operative incisions. Likewise, patients with mixed arterial and venous disease should be evaluated to insure adequate arterial supply for wound healing. Unusual varices such as those occurring in young patients, unilateral lesions, or those in atypical locations should be studied with venography to identify lesions such as arteriovenous fistulas, deep venous occlusion, and agenesis of valves.

Attention to the details of the operative technique will diminish intraoperative complications. Blood loss can be reduced by placing the operating table in a Trendelenburg position, thereby diminishing venous pressure. One author[14] has suggested elevating the extremities on a slotted rack device at a 30 degree angle to further decrease venous pressure and increase circumferential exposure.

In 1916, Homans[43] recommended ligation of the greater saphenous vein proximal to its tributaries at the level of the femoral vein. Knowledge of the anatomy and relationship of the saphenous and femoral veins and the femoral artery is important to this procedure. Lack of anatomic orientation has led to major injuries to the femoral vessels. The true incidence of these injuries is unknown, but they are thought to be more common than the infrequent cases reported in the literature.

Most arterial injuries are related to the failure to identify the normal variations of superficial external pudendal artery in relation to the

femorosaphenous junction.[17] Profuse bleeding occurs when this artery is not properly identified and further damage may occur if blind clamping is used. Damage to major arteries is rare. Eger[22] reported a case of inadvertent stripping of the superficial femoral, popliteal, and posterior tibial arteries with successful vascular reconstruction utilizing a composite graft to the peroneal artery. Liddicoat[55] reported two cases of arterial insufficiency following venous procedures. Both patients had surgical interruption of the femoral arterial system requiring reconstruction. This author emphasized the importance of early recognition and repair to prevent permanent deficits. Likewise, injuries to the femoral vein are rare. Haeger and Sandburg[38] reported bleeding from a rent in the femoral vein during varicose vein surgery in six of 2000 patients. This type of injury is repaired by lateral suture without sequelae. Transection of the femoral vein is a more serious problem. Femoral vein interruption produces signs of acute thrombosis and will result in chronic venous insufficiency with severe disability if not repaired. Tera[86] reported a case of femoral vein repair without sequelae by using an interpositioned saphenous vein graft taken from the opposite extremity.

Although early postoperative edema is common, permanent edema of the lower extremities following surgery for varicose veins is a rare postoperative problem. Permanent edema may result from lymphatic disruption at the time of venous surgery. Lymphatic vessels of the lower limb are divided into two groups: superficial and deep. The superficial lymphatic vessels lie in the subcutaneous fat in a course along the greater and lesser saphenous veins. Because of the proximity of these lymphatics to the long saphenous vein, it is surprising that lymphatic disruption with subsequent edema is not a more common problem.[22]

In a preliminary study, lymphatic disruption was demonstrated by lymphangiography in patients undergoing vein stripping (Fig. 4).[91] When lymphatic disruption is extensive, permanent lymphedema may result. Prevention must be directed at the use of a small acorn-tip for venous stripping to limit the degree of perisaphenous lymphatic disruption and stripping from groin to ankle in a downward fashion.

Sensory disturbances in the distribution of the saphenous and sural nerves are recognized complications of varicose vein surgery and may result in annoying symptoms. The saphenous nerve, which supplies the skin of the medial leg, exits from the adductor canal and becomes subcutaneous on the medial side of the knee between the tendons of the sartorius and gracilis. The saphenous nerve then accompanies the saphenous vein to the ankle. Injury to this nerve can usually be avoided by careful dissection of the nerve from the saphenous vein at the ankle and vein stripping with a small acorn-tip. Wellwood and his colleagues[93] studied 30 patients who had bilateral saphenous vein stripping; one leg stripped up and one leg stripped down. There was a significantly greater incidence of sensory impairment in those legs stripped from ankle to groin. The authors proposed that the head of the stripper engages an inverted "v" formed by the saphenous vein, avulsing a segment of nerve passing through this "v." Garnjobst[28] reported four cases of permanent saphenous nerve deficit which occurred with secondary

incisions at the knee. This author suggested that the greatest damage occurs from blind dissection in this area. Blind grasping with a hemostat during the course of subcutaneous dissection in this area should be particularly avoided.

The sural nerve accompanies the lesser saphenous vein to the level of the lateral malleolus and supplies sensation to the posterior leg and lateral foot. This nerve likewise can be injured during removal of the lesser saphenous vein. Most sensory loss is transient and may even go unnoticed by the patient. The most common area of decreased sensation is the medial malleolus. Permanent disabilities are rare but may result in formation of painful neuromas.

Injection and compression therapy for varicose veins, though common in Europe, is seldom practiced in this country. However, the possibility of local damage as a result of extravasation of sclerosing solution should be kept in mind. Injuries such as skin necrosis, hematoma, infection or even local hirsutism have been reported.[58] Accidental intra-arterial injection during sclerotherapy may cause gangrene leading to limb loss.[24]

Figure 4. Lymphangiogram in a patient who had vein stripping for varicose veins. Lymphatic disruption is seen in the medial saphenous group. (Reproduced from van Bellen, B., Gross, W. S., Verta, M. J., Jr., et al.: Lymphatic disruption in varicose vein surgery. Surgery, 82:257, 1977. Used with permission.)

Phlebography is seldom needed in the evaluation of varicose veins, except in children or young adults (less than 20 years of age), or in patients with unusual or recurrent varicosities in whom visualization of the deep venous system is deemed necessary. Varicose veins may present as part of the clinical picture of congenital hemangioma.[21] If associated congenital arteriovenous malformation is suspected, an arteriogram should be done to further evaluate the condition. Stripping of varicose veins in multiple small arteriovenous fistulas is not advisable; however, in patients with local anomalous connections to the deep venous system which produce varicose veins, ligation of the communicators is helpful.

VENOUS INJURIES

Venous injuries are frequently associated with major arterial trauma. In penetrating wounds, injuries to the venous system especially the vena cava may cause profuse bleeding, leading to death if prompt surgical intervention is not instituted. Apart from penetrating wounds, the diagnostic or therapeutic procedures which use the vein as an entry for placement of the catheter, cardiac procedures, and vena cava filters have introduced a wide range of new problems. Infusion phlebitis, septic thrombophlebitis, or bacteremia from arm veins is not infrequently encountered when patients receive prolonged infusions or during administration of hyperalimentation. Misplacement of catheters or embolization of the catheter may present challenging problems. Percutaneous catheterization of the subclavian vein, a common procedure for monitoring of central venous pressure, may cause complications including pneumothorax, hydrothorax, arteriovenous fistula formation, and nerve damage. Fatal complications such as cardiac tamponade have also been reported.[19]

Venous thrombosis and pulmonary embolism may follow venous injury. The incidence of these complications is rare. However, they appear to be more common after ligation than venous repair. Gasper[29] reported three cases of thrombosis following 27 vein ligations but there was no thrombosis following ten direct vein repairs. Others[74] have also reported an extremely low incidence of pulmonary embolism following surgical repair.

Management of major venous injury evolved over the last 20 years from simple ligation to venous repair and reconstruction. Experience with vascular injuries during the Viet Nam War led to a trend away from venous ligation.[74] In the Viet Nam series, 37.7 per cent of arterial injuries were associated with major venous injuries. The management of these venous injuries may influence the success of the arterial reconstruction, especially at the popliteal level. In a series of 125 patients requiring amputation following popliteal artery injury, severe postoperative edema as a result of venous ligation contributed to the need for amputation.[75] In an animal study, Barcia[3] demonstrated that ligation of the

common femoral vein causes a decrease in arterial flow, further supporting the influence of venous outflow on the success of arterial reconstruction. These clinical and experimental observations have called for direct repair of venous injuries. In dealing with vascular injuries of the leg, efforts must be made to provide autogenous graft material to maintain arterial and venous continuity. When saphenous vein is needed for arterial and venous reconstruction, it must be taken from the opposite extremity in order to maintain venous collateral flow on the injured side.

Thrombosis of the repaired venous segment is the major complication of venous reconstruction. However, even short periods of patency may allow for the development of venous collaterals to prevent acute venous insufficiency in the lower extremity.[3] Anticoagulation with heparin has not been demonstrated to decrease the rate of thrombosis in grafts in the venous system.[3] The creation of a distal arteriovenous fistula as an adjunctive measure to increase flow and pressure through the reconstructed venous segment has been suggested.[52] The value of this approach needs further experience to determine its efficacy in maintaining venous patency in patients with venous trauma. When interposition grafts are required for venous repair, the use of autogenous vein is preferred. If the caliber of the vein is too small, a composite autogenous graft may be constructed. Synthetic graft material has a poor patency rate in the venous system. Moreover, thrombosis of an autogenous graft in the venous system may be followed by recanalization. Synthetic grafts do not share this ability to recanalize.[39]

Prevention of catheter injuries is by careful placement of the catheter with radiographic control to insure the exact location and the absence of pneumo- or hydrothorax. Infection must be treated promptly by systemic antibiotics and heparin, followed by removal of the catheter. Any catheter placed in the venous system for more than 48 hours should be cultured. Retrieval of a loose catheter embolized to a particular segment may present a problem. This can be accomplished by use of the basket catheter commonly employed by urologists to extract stones. Such a maneuver, however, should be done under a radiographic monitor.

Other forms of venous injury which have increased in frequency recently involve drug abuse. The use of a nonsterile needle often introduces infection, causing septic thrombophlebitis. In addition, the hypertonicity of the mixing solution and the powder content of various drugs may cause severe intimal damage, resulting in thrombus or distal embolization. The lack of anatomic knowledge of the drug user in performing needle puncture often leads to formation of an infected false aneurysm or arteriovenous fistula at the site of entry. Infected false aneurysms may cause fatal complications such as bacteremia or bleeding. The treatment of choice is by systemic antibiotics and surgical attempt to isolate the infected artery from the circulation. This is achieved by proximal and distal ligation of the aneurysms with arterial flow supplied via an extra-anatomical route through a sterile field. In infected femoral aneurysms, this can be handled effectively by the use of obturation bypass.[37]

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Biliary Tract Surgery

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As surgeons, we would like to think that surgery on the extrahepatic biliary tree has progressed significantly since the first planned gallbladder operation, a cholecystostomy performed by J. Marion Sims in 1878. This patient succumbed to postoperative hemorrhage. However, despite the fact that most surgeons have an extensive experience with these types of operations, morbidity remains high and mortality rates significant. Much of this morbidity is caused by factors common to all surgical procedures, namely the complications wrought by advancing age, atherosclerotic cardiovascular disease, pulmonary insufficiency, diabetes mellitus, and the like. However, many complications following biliary tract operations are unique to this type of surgery and have their origins in judgmental or technical errors. They are therefore in many cases preventable. It is with this group of complications that this article will deal.

Subhepatic Accumulations and Abscesses

Following cholecystectomy, depending upon the state of inflammation in the gallbladder and the skill of the operator, there will be a certain amount of drainage of bile, blood, lymph, and peritoneal fluid into the subhepatic space. In the majority of instances, this is of no clinical importance. Bile and subhepatic collections can originate from many sources. Damage to the liver or the gallbladder bed may rupture a small ductule. Accessory ductules not infrequently drain into the gallbladder through the gallbladder bed, and if these are not occluded, bile leakage can result. A ligature may slip off the transected cystic duct, or a choledochotomy closure may leak.

Because of these accumulations and the much less frequent bile leaks and fistulas, routine drainage of the right upper quadrant is advocated utilizing a Penrose drain. If no significant bile drainage occurs, these drains may be removed within 48 hours, and it has been shown that this leads to no increased incidence of infectious complications.[40] Even those who oppose routine drainage after cholecystectomy still use drains for acute cholecystitis or significantly debilitated or immunosup-

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pressed patients.[32] However, it seems to us that routine drainage for a short period of time postoperatively is the preferable path, as surely in some cases reoperation will be prevented by the presence of the drain allowing egress of fluid outside the abdomen.

Wound Infection

Only when surgeons cease regarding the presence of a wound infection as a "minor complication" will any significant headway be made in lowering the incidence of wound infections on general surgical services. An excellent prospective study of wound infections was carried out by Cruse at the Foothills Hospital in Calgary, Alberta. A wound infection rate of 2 per cent was noted with cholecystectomy alone, and this rate was not significantly different when operative cholangiography was performed along with the cholecystectomy. However, when a common bile duct exploration was carried out, the rate of infection was 7.9 per cent. Of even greater interest in this study was the wide difference in wound infection rates noted by different surgeons.[10] It has been clearly shown by the experimental work of Condie and Ferguson that in the absence of deliberate faults in surgical technique, even heavily contaminated wounds will heal normally.[9] Therefore, it is incumbent upon the surgeon to take all necessary steps to avoid excessive trauma to the wound. These include the proper length and location of incision, so that undue traction is not necessary on the wound edges in order to obtain exposure. A right subcostal incision is preferable in most cases. A plastic sheet adherent to the entire abdominal wall may decrease some of the surface contamination, and a ringed plastic wound protector can be inserted into the wound and will prevent endogenous contamination and keep the wound from drying out during the procedure.[11, 12]

In addition, it is necessary to prevent the formation of dead space by a clean perpendicular incision through all tissue layers, to obtain absolute hemostasis in the wound and to avoid excessive suture material or electrocautery in the wound. The choice of suture for fascial closure should be from among the available monofilament or absorbable suture materials. Such material as running 0 polyglycolic acid, 0 polypropylene, or interrupted monofilament wire sutures are all acceptable. For closure of the skin, the least trauma is afforded by skin tapes which are accurately placed after benzoin has been applied to the wound edges.

Hemorrhage

Hemorrhage during and after cholecystectomy continues to be a source of morbidity. Significant intraoperative hemorrhage generally occurs from the cystic artery or the right hepatic artery which has been inadvertently injured. The danger here is generally not the hemorrhage itself but rather the ill-advised steps which may be taken to stop it. It is essential that no blind clamping be carried out in this area, as injury to the bile ducts may result. If hemorrhage cannot be controlled with simple pressure and suction, the assistant may put his finger in the foramen of Winslow and digitally occlude the hepatic artery inflow, thus

allowing accurate hemostasis to be achieved.

Another source of intraoperative hemorrhage is a subcapsular hematoma of the liver, generally brought about by ill-placed or too vigorous retraction. A small hematoma which is not expanding requires no treatment. However, a larger hematoma or one which appears to be growing should be unroofed by incising the liver capsule with suture ligation of the offending vessel. If this is not done, massive subcapsular hematomas can result with serious sequelae.[22] The last source of bleeding is from the gallbladder bed itself. A generation of surgeons has been educated to close the gallbladder bed in order to stop bleeding and decrease the number of postoperative adhesions. Both of these concepts are in error. Rather than stopping bleeding, closure of the gallbladder bed merely hides the bleeding and allows it to collect in the subhepatic space postoperatively. In addition to this, there is no evidence that closure of the gallbladder bed reduces postoperative adhesions. Hemostasis should be achieved with electrocautery at the conclusion of cholecystectomy, so that the gallbladder bed is absolutely dry. Following this, closure of the bed is not indicated.

Any of the above mentioned causes of bleeding may give rise to postoperative hemorrhage, but in addition, the drain site may be a source of bleeding, and this can be mistaken for exiting intraperitoneal blood. This problem can be avoided by incising only the skin over the drain site and not making a deeper cut with the scalpel. A hemostat may then be pushed through the subcutaneous tissues and muscle to form the drain site. Another error which can be made is the assumption that a soft drain will always allow exit of the blood in the case of hemorrhage. This is not necessarily so, as large amounts of clotted blood can collect in the subhepatic space with relatively modest drainage. Finally a word of caution regarding biliary tract surgery on the chronically jaundiced patient. In some of these cases, a bleeding disorder will be present manifested by elevated prothrombin time, and this should be corrected with supplemental vitamin K prior to operation.

Bile Leak and Fistula

The sequelae of bile spillage into the abdomen are quite unpredictable. Even a small amount of bile may give rise to extreme toxicity with evidence of peritonitis, whereas massive bile accumulations and bile ascites can occur with virtually no local or systemic signs except abdominal swelling.[31] Providing a drain is in place, the bile will generally exit from the subhepatic space and the problem will be a self-limiting one. The management of a patient with bile leak will depend upon the patient's response to the leakage and also the etiology of the leak. Obviously, in a patient with a common duct injury associated with a bile fistula or a patient with retained stones in the common bile duct associated with a disrupted cystic duct stump, reoperation will be mandatory. In general, if the patient is tolerating the leakage without evidence of peritonitis or significant systemic toxicity, the drain may be left in place with a colostomy bag over the drain site. In the majority of cases unassociated with distal obstruction or common duct injury the fistula will gradually

close without serious sequelae. After 7 to 10 days, if the fistula output is not diminishing, radiologic investigation should be carried out. Probably the simplest method is to replace the Penrose drain with a small rubber catheter and then inject the catheter with water-soluble contrast material. If this sinus tract injection does not delineate the bile ducts, an intravenous cholangiogram can be attempted. Usually, these two methods will provide all the information that is needed. However, if additional studies are required, either skinny-needle transhepatic cholangiography or endoscopic retrograde cholangiography (ERCP) may be attempted. If no mechanical cause is found, it can be assumed that the majority of these leaks will heal spontaneously. In the rare case in which significant quantities of the normal 1000 to 1500 ml. daily bile output are being diverted out the fistula, bile refeeding via a pediatric feeding tube can be accomplished with resultant beneficial effects to the patient.

Postoperative Jaundice

Jaundice following biliary tract surgery is an uncommon but greatly feared complication, as its presence always raises the spectre of a bile duct injury. Transfusion reactions, hepatitis, and other medical forms of jaundice will not be dealt with here. Retained common duct stones with or without ascending cholangitis probably represent the commonest surgical problem, although an overlooked periampullary tumor, stenosis of the sphincter of Oddi, or papillitis may be present and represent the etiology of the biliary tract disorder.[1, 34] In addition, a ligated hepatic artery may uncommonly yield hepatic necrosis and jaundice.

The main problem then is that of overlooked or recurrent biliary tract calculi. Glenn, in an excellent review of 16,700 patients undergoing cholecystectomy, indicated that common bile duct exploration was performed in 24 per cent of these patients, and that in nearly half, calculi were removed. The incidence of residual calculi was 1.1 per cent of those undergoing cholecystectomy and 4.3 per cent for those undergoing cholecystectomy and common duct exploration.[16]

It is obvious then that the surgeon performing biliary tract surgery must be alert to the possibility of common duct stones being present, even though these may be asymptomatic. It is well recognized that the incidence of common bile duct stones is higher with advancing age and also when the operation is performed for acute cholecystitis.[38] The recent availability of expedient operative cholangiograms has definitely decreased the incidence of overlooked stones and the incidence of negative common duct explorations. However, many technical details of cholangiography such as dye concentration, amount of dye used, and cessation of respiration during the cholangiogram must be adhered to if the cholangiogram is to be reliable in indicating the presence or absence of stones.[19, 21–23]

In addition to routine operative cholangiography in the demonstration of common duct stones, certain clinical criteria for common duct exploration should be adhered to. These are a dilated cystic or common bile duct, palpable stones in the common bile duct, and a history of jaundice or pancreatitis.[3, 8] The presence of small stones in the gallblad-

der is a less valuable indicator of the presence of common duct stones and should not serve as the sole indication for common bile duct exploration.

Despite the opinion that usage of a T-tube following choledochotomy is a fetish,[7] there are certain advantages to their routine usage. An operative T-tube cholangiogram can be taken following closure of the common duct, and this in some cases will disclose overlooked stones. In addition, if cholangiography is performed before tube removal on about the 7th to 10th postoperative day, stones may be visualized, and various nonoperative means utilized to effect their removal. Foremost among these are dissolution of stones with various solutions and direct manipulations via the T-tube tract. In recent years, the use of high doses of heparin instilled through the T-tube has been successful in dissolving stones,[14] and manipulation through the T-tube site with a stone grasping basket is likewise becoming a commonly used and very successful means of extracting stones.[6, 27] The case history of Z. J. (below) illustrates a successful outcome following heparin instillation for a retained common duct stone. In general, utilization of clinical and operative criteria for the presence of common duct stones along with good quality operative cholangiograms and meticulous common bile duct exploration should minimize the problem of recurrent and retained common duct stones.

CASE NO. 1. Z.J. is a 47-year-old female who was admitted to the Loyola University Medical Center on August 15, 1976 with a 2-week history of right upper quadrant pain. She was febrile and jaundiced, and a diagnosis of acute cholecystitis and ascending cholangitis was made. She was operated upon promptly and a markedly inflamed gallbladder was removed. Common bile duct exploration disclosed numerous stones ranging up to 0.5 cm in diameter. Following common bile duct exploration, a completion operative cholangiogram was normal. However on the 7th postoperative day, a subsequent T-tube cholangiogram disclosed a stone in the distal common duct (Fig. 1). She was begun on a constant heparin infusion via the T-tube, consisting of 75,000 units of heparin in 1 liter of saline given daily. After 8 days, there was no stone visible on repeat T-tube cholangiogram and the T-tube was removed (Fig. 2). She has remained asymptomatic with normal liver function tests since that time.

Postoperative Pancreatitis

Acute pancreatitis can be seen in the postoperative period after any general surgical procedure but seems to be especially common after operations on the extrahepatic biliary tree. There is general agreement that when the disease occurs in the postoperative period, it is associated with excessive mortality rates ranging from 25 to 42 per cent.[2, 28, 39] The addition of operative cholangiography to cholecystectomy does not appear to increase the incidence of postoperative pancreatitis,[22, 38] but common bile duct exploration definitely increases the incidence. In a study of 3257 patients undergoing cholecystectomy, Boquist encountered 29 deaths, and over one-half of these were from acute postoperative pancreatitis.[5] It appears that local trauma plays a role in the etiology of postoperative pancreatitis, but the method of common duct exploration undoubtedly is important. Despite considerable evidence that forceful dilatation of the ampulla with dilators greater than a No. 3 Bakes size is

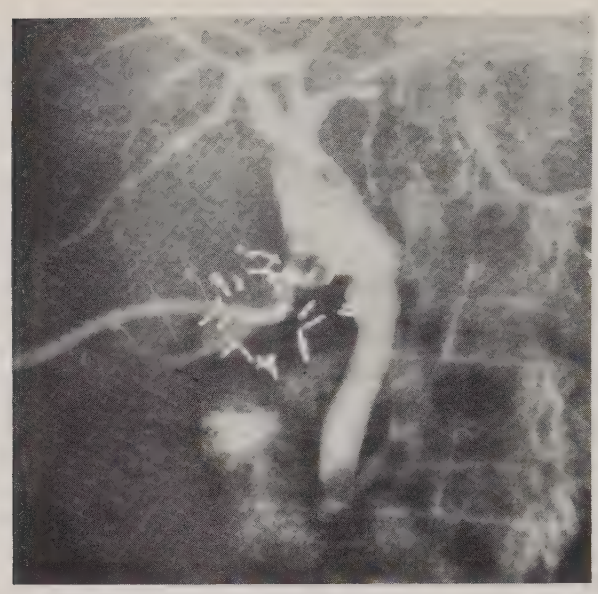

Figure 1. T-tube cholangiogram demonstrating a 1 cm stone in the distal common bile duct.

harmful and leads to fibrosis and narrowing,[30] many surgeons still insist upon forcibly dilating the ampulla at the time of choledochotomy.

The difficulty in making the diagnosis of acute postoperative pancreatitis is well known. The importance of elevated serum and urine amylase levels is obvious in this situation. Once the diagnosis is made, very aggressive treatment must be instituted including nasogastric suction, vigorous colloid and crystalloid replacement, and early operation if the patient is deteriorating on nonoperative management. The use of anticholinergic and antibiotic drugs should likewise be considered early despite the fact that evidence of their effectiveness in aborting an attack is controversial. If operation is necessary due to a rapidly deteriorating clinical state, extensive sump tube drainage of the pancreas is probably indicated as is a decompression gastrostomy. Provision for long-term hyperalimentation can be accomplished with either a feeding jejunostomy or postoperative intravenous hyperalimentation.[24]

Injuries to Structures in the Porta Hepatis

Injury to the bile ducts is probably the single most serious and feared complication of biliary tract surgery. In a review of 63,252 cholecystectomies performed in the central United States, Hermann noted 336 bile duct injuries, an incidence of approximately 0.5 per cent.[20] There are many ways in which the bile ducts can be injured during cholecystectomy, but certain patterns are repetitive. Oftimes during cholecystectomy, bleeding is encountered in the porta hepatis, and blind attempts are made to clamp or suture ligate the offending vessel with-

out adequately delineating the anatomy. Nor is the bloodless or "easy" cholecystectomy free of problems. Traction may be exerted upon the gallbladder, so that the cystic duct ligation actually impinges upon the bile duct, causing compromise of its lumen. A third cause of bile duct injury results from the ignorance of anomalies of the structures at the porta hepatis. These are quite frequent and if a careful dissection is not carried out in this area, damage to the bile ducts and branches of the hepatic artery may occur.

If a bile duct injury is noted at operation, the preferred management includes primary repair of the defect with stenting of the common bile duct by a T-tube introduced into the duct through a separate opening. However, often the first sign of bile duct injury is a postoperative bile leakage or a late stricture manifested by jaundice. In a review of 958 patients with bile duct injuries seen at the Leahy Clinic, 915 followed cholecystectomy, and it was frequently noted that there was either a poor choice or performance of an incision leading to poor exposure in many of these patients.[36] The surgeon faced with the formidable task of

Figure 2 T-tube cholangiogram performed 8 days after instillation of heparin solution into T-tube. No residual stone is noted, and there is free reflux into the pancreatic duct.

re-establishing continuity between the biliary tree and the gastrointestinal tract must obtain as much information as possible preoperatively. A copy of the previous operative report can be very helpful in this regard. If a bile fistula exists, injection of contrast material can delineate the ductal injury. Otherwise, skinny-needle cholangiography or endoscopic retrograde cholangiography can be carried out. One of the major problems at operation is the identification of the proximal and distal ends of the bile ducts. The actual reconstruction depends upon whether the two ends can be approximated. If so, a primary anastomosis with a splinting T-tube can be carried out. Where a segment of bile duct is missing rendering direct anastomosis impossible, either a choledochoduodenostomy or a Roux-Y choledochojejunostomy must be carried out. Although controversy exists on the routine use of internal stents, many surgeons with extensive experience advocate their use for prolonged periods of time postoperatively.[36]

Hepatic artery injuries probably occur far more commonly than is recognized. In the past, mainly on the basis of experiments utilizing dogs, it was felt that hepatic artery ligation would be uniformly fatal in the human. However, recent evidence has disclosed that the hepatic artery in man is not an end artery, and rapid collateralization occurs regardless of the level of ligation, the liver being sustained by the new collaterals and also by portal venous blood.[4, 26] However, despite this reassurance, it would seem wise at the time of cholecystectomy to attempt arterial repair on any hepatic artery which is damaged. Obviously the avoidance of bile duct and hepatic artery injuries will depend upon the skill of the surgeon. There is, however, an alternative to the standard method of cholecystectomy which is probably safer over the long run than the usual method of preliminary dissection of the triangle of Calot followed by ligation of the cystic artery and duct and removal of the gallbladder. This method is retrograde removal of the gallbladder from the fundus of the gallbladder to the porta without preliminary identification of the structures at the porta hepatis.[20] Safe cholecystectomy will depend upon an adequately placed incision of sufficient length to give access to the gallbladder. The dissection is begun at the fundus of the gallbladder where the subserosal plane is entered and developed along both sides of the gallbladder down to the porta. Any small vessels are cauterized as the dissection proceeds, and no attempt is made to delineate cystic or common bile ducts. Following this, the gallbladder is removed from its bed until it is attached only by the cystic duct. Any arterial branch going into the gallbladder during this phase of the dissection can be assumed to be a cystic artery and can be hemoclipped with impunity. At this point, the junction of the cystic and common hepatic ducts is delineated by sharp and blunt dissection and a cystic duct operative cholangiogram is taken. If this verifies normal anatomy, the cystic duct is ligated and the gallbladder removed. Using this technique, the common variations of bile duct anatomy should be easily managed[29] and bile duct injury should be an extreme rarity. Absolute hemostasis of the gallbladder bed and porta hepatis should be obtained with judicious use of the electrocautery, following which the gallbladder bed should be

left open. The right upper quadrant should be liberally irrigated with saline solution, not only to detect further bleeding but also to dilute any contamination or blood which may remain. After this, fascia should be approximated with monofilament suture material and skin tapes applied.

Complications Secondary to the Use of Drainage Tubes

In the early postoperative period, T-tubes can stop draining due to obstruction by inspissated bile or blood clots. Failure to recognize this can lead to leakage from the choledochotomy site. Therefore it is essential to irrigate any T-tube with a sterile saline solution if it is not draining satisfactorily. Another frequent occurrence in the postoperative period is dislodgment of the T-tube from the common bile duct. This is often brought about by leaving insufficient slack inside the abdomen so that when the patient arises from the supine to the upright position, the tube, being fixed at the skin, is pulled out of the bile duct. Obviously, the shorter the time from operation, the more potentially serious this problem may be, as bile peritonitis can occur in the absence of a walled-off tract around the T-tube. Treatment in this situation consists of leaving the T-tube in place as long as it continues to drain and then removing it following cessation of bile drainage.

Less commonly, trouble is encountered removing the T-tube from the common bile duct. This generally results from a suture passing through the common duct and the T-tube, but it also may result from a very tight closure of the common duct around the tube. This latter problem can be avoided by cutting a wedge out of the T-tube opposite the exiting arm of the tube. This will enable the tube to buckle on itself upon withdrawal. Gentle continuous traction will generally lead to the removal of a retained tube. If traction is placed on the T-tube and a hemostat applied at skin level, this constant but gentle traction will generally lead to expulsion of the tube in time. These potential problems are quite uncommon, and the advantages of having a T-tube such as postoperative cholangiography and irrigation or manipulation of retained stones far outweigh the potential disadvantages.

Failure of Cholecystostomy

There is little controversy regarding the role of cholecystectomy as the operation for the definitive management of calculous gallbladder disease. However, the operation of cholecystostomy enjoys periodic waves of enthusiasm as a safer and perhaps even definitive operation in the management of acute cholecystitis. Few surgeons would disagree that there are certain circumstances in which cholecystostomy may be the preferred treatment. Occasionally, at the time of planned cholecystectomy, severe portal hypertension is encountered, making the removal of the gallbladder an exceptionally difficult undertaking. In addition to this, such severe inflammation may be present that the porta hepatis cannot be dissected safely. In either of these instances, cholecystostomy may prove lifesaving. However, the main advantage of cholecystostomy should result from its preoperative planned usage in extremely critically

ill patients with acute cholecystitis, as the operation can be carried out under local anesthesia quite readily.[35] If general anesthesia has been decided upon, and the decision for cholecystostomy is an operative one, much of its advantage is lost. In addition, it is critical to note that in the presence of ascending cholangitis manifested by right upper quadrant pain, fever, and jaundice, cholecystostomy cannot be depended upon to yield satisfactory decompression of the biliary tree. In this instance, only T-tube drainage of the common bile duct will suffice.[13, 33]

There is a certain degree of optimism among surgeons for cholecystostomy in the management of acute cholecystitis.[17] However, many series of patients have been reported following cholecystostomy which indicate that this operation is not definitive in many cases. Malstrom found twelve deaths in his series of 63 cholecystostomies, and nine of these were from local complications of the cholecystostomy such as peritonitis and abscess formation. Nine of the survivors required subsequent closure of the chronic bile fistula, and in only 31 per cent was the cholecystostomy definitive. The remainder either died, required subsequent cholecystectomy or lived with symptomatic gallbladder disease.[25] Others have found that the outlook is somewhat better following cholecystostomy, especially if the postoperative cholangiogram discloses no stones in the gallbladder or biliary tree. Over half of these patients can be expected to remain symptom-free during lengthy follow up periods.[15, 37]

Our dissatisfaction with cholecystostomy stems from the above considerations and also from a series of five consecutive patients in whom cholecystostomy was carried out for acute cholecystitis and in whom cholecystectomy had to be subsequently performed, in some cases within several months of the cholecystostomy. The case history of such a patient is given below.

CASE No. 2. P.E. is a 56-year-old male and was admitted to the Loyola Medical Center in April 1976 for cardiac catheterization. He had a history of a prior myocardial infarction in November 1975. Catheterization disclosed a 90 per cent proximal occlusion of the left anterior descending coronary artery, and the patient was scheduled to undergo coronary revascularization. At this time, however, he suffered the acute onset of right upper quadrant pain, fever, and leukocytosis. This failed to respond to medical management, and on May 10, 1976, he underwent cholecystostomy under local anesthesia. At that time, the gallbladder was perforated with approximately 200 ml of bile in the subhepatic space. A postoperative cholangiogram disclosed free filling of the cystic and common bile ducts with no evidence of stones in the gallbladder or ductal system (Fig. 3). Eight days postoperatively, he began to have fever and chills. At this time, the cholecystostomy was draining normally, and his symptoms receded with antibiotic treatment.

It was planned to discharge him from the hospital for approximately 3 months, as he had developed heparin-induced platelet antibodies, and it was felt that cardiopulmonary bypass was impossible at this time. However, he was readmitted on July 6, 1976 with fever, chills, and right upper quadrant pain. Again the tube was draining freely. At this time, his temperature was 38.5° C. White blood cell count was 5200 cells per cu mm. All blood cultures were negative, but bile cultured Pseudomonas aeruginosa, Proteus mirabilis, Escherichia coli, and Klebsiella pneumoniae. He was treated with ampicillin with complete resolution of symptoms within 1 day. On July 14, 1976, he underwent cholecystectomy under general anesthesia. His operative and postoperative course was smooth,

and no EKG changes were noted postoperatively. There has been no further evidence of right upper quadrant pain, fever, or chills, and he has subsequently undergone a successful coronary revascularization.

Late Complications Following Cholecystectomy

Unfortunately, not every patient who undergoes cholecystectomy is permanently relieved of his symptoms. A wide variety of pathologic entities can account for persistent or recurrent symptoms. These include bile duct stricture with or without ascending cholangitis, recurrent or retained common duct stones, a gallbladder remnant or long cystic duct remnant, or a painful neuroma in the porta hepatis.

Glenn and McSherry described 253 cases out of 5859 patients undergoing cholecystectomy who had "postcholecystectomy syndrome" and required a second operation. Two-thirds of these patients had diseases of the liver, bile ducts, or pancreas, such as a retained cystic duct stump, common bile duct stones, stenosis of the ampulla, or pancreatitis. One-third however had disease outside of the biliary tree which had been previously unrecognized, such as duodenal ulcer or hiatus hernia.[18]

The management of patients with "postcholecystectomy syndrome" must be individualized. Surely if jaundice is present, a skinny-needle transhepatic cholangiogram or endoscopic retrograde cholangiogram

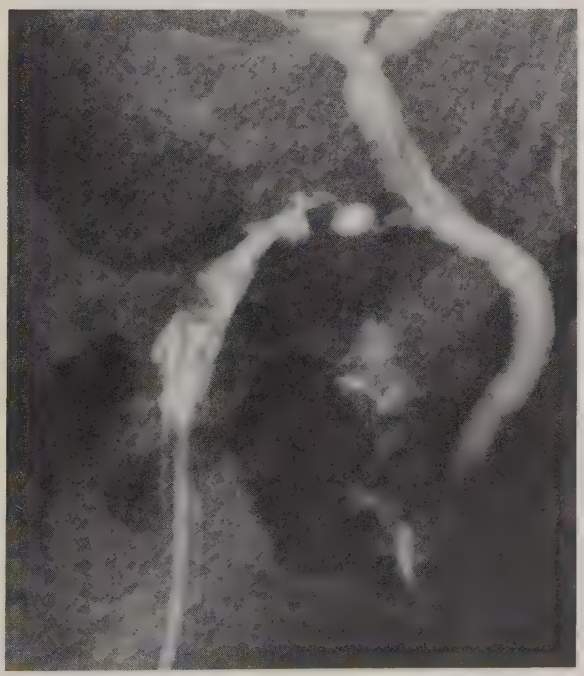

Figure 3. Injection of contrast material into the shrunken gallbladder via a cholecystostomy tube demonstrates no stones and free passage of dye from the gallbladder into the bile duct and duodenum.

will be required. Reoperation will depend upon demonstration of actual pathology, except in the quite rare case of a painful neuroma, whose presence can only be inferred from the patient's history and not demonstrated by any currently available tests. With careful adherence to accepted indications for cholecystectomy along with a technically competent removal of the gallbladder and careful exploration of the bile ducts if indicated, all of these late syndromes should be seen infrequently. The case history of patient J.L. (below) represents a recent instance of postcholecystectomy syndrome managed successfully.

CASE NO. 3. J.L. is a 45-year-old woman who underwent an uncomplicated cholecystectomy 20 years prior to her admission to the Loyola Medical Center. She presented here with a 2-month history of epigastric pain radiating around the right ribs to the right scapula. This pain had no relationship to meals and was not associated with vomiting. There were no particular food intolerances, and she had not noticed any weight loss. Physical examination was normal. A CBC, SMA12, and serum amylase were normal. An upper gastrointestinal x-ray and intravenous cholangiogram were likewise normal. A superior mesenteric and celiac angiogram was normal, and ERCP disclosed a nondilated common bile duct without stones but a remarkably elongated, dilated, and tortuous cystic duct (Fig. 4). The patient underwent laparotomy and excision of the long cystic duct remnant which contained no stones. A common bile duct exploration and operative cholangiogram were normal, and she has been totally asymptomatic since that time.

Figure 4. ERCP disclosing retrograde injection of dye into a normal common bile duct and pancreatic duct. Arrows outline the markedly elongated, dilated, and tortuous cystic duct remnant.

CONCLUSIONS

Today, many of the foregoing complications of biliary tract surgery are seen in elderly patients who also possess significant cardiopulmonary deficits, diabetes mellitus and other disorders of advancing age. Operations in this group of patients could in many cases be avoided by the decision for earlier cholecystectomy in younger patients with symptomatic and asymptomatic gallstones. In addition, the earlier operative treatment of acute cholecystitis rather than a mandatory period of medical management followed by operation only for those who fail to improve would likely decrease the number of total complications. The judicious use of antibiotics in the elderly and in those patients with diabetes mellitus and acute cholecystitis would likewise bring about a decreased incidence of septic complications. The technical aspects of cholecystectomy need constant reinforcement. Cholecystectomy from the fundus downward to the porta has proved fast and extraordinarily safe, with an absolute minimum of risk to the structures in the porta hepatis. Operative cholangiography should be routine during cholecystectomy and with good quality cholangiograms, the incidence of negative common bile duct explorations should be decreased. This too will lessen subsequent morbidity and mortality. Fortified with this knowledge, the would-be biliary tract surgeon is far less likely to encounter major surgical complications.

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Operations for Inflammatory Bowel Disease

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Patients suffering inflammatory bowel disease undergo operation either to cure the disease (ulcerative colitis) or to eliminate the complications of the disease (ulcerative colitis, Crohn's disease). These patients are a risk not only for the complications inherent to any major intra-abdominal operation, but also for certain complications specific to their disease and operative procedure.

WOUND COMPLICATIONS

Incision

The placement of the abdominal incision should be made to afford ready access to the organs to be manipulated, yet must resist dehiscence, evisceration, and hernia. Operations upon the diseased bowel may carry the surgeon from the duodenum to the splenic flexure and the deep pelvis. We specifically condemn the longitudinal incision as being inadequate to visualize with ease all portions of the abdominal and pelvic cavity, prone to herniation and separation, and, if infected, leads to difficult ventral hernias.[42] We prefer for *all* operations a horizontal or transverse infraumbilical incision usually extending from one anterior axillary line to the other.[38]

Infection

Cole and Bernard[11] indicate that 14 per cent of wounds through which the intestine is opened become infected. Less than 4 per cent of patients specifically operated upon for inflammatory bowel disease require rehospitalization for wound complications.[39]

The commonest site of wound infection is in the subcutaneous tissue. The frequency of such sources of gross contamination as fistulae,

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intra-abdominal abscesses, as well as the inevitable contamination associated with transection of the bowel, would seem to make such infections inevitable. However, while contamination cannot be eliminated, the contaminated wound may be prevented from developing a frank abscess by a number of means. Careful technique and the avoidance of dead space are paramount.

When frankly contaminated regions are in juxtaposition to the wound, or if there is gross fecal contamination of the wound, the prudent surgeon will elect to delay or not to close the superficial wound and subcutaneous tissue. The employment of this technique will prevent almost all true wound infections, decrease postoperative hospitalization, and give a pleasing cosmetic result. Bacteroides, Aerobacter, streptococci, and staphylococci are the most common offenders. If a wound abscess is suspected, the wound should be widely opened, the exudate cultured, and the wound copiously irrigated and lightly packed with a fine mesh gauze. Frequent changes of packing and irrigation will result in healthy wound granulations within 4 to 5 days; at this time the surgeon can elect either secondary closure at the bedside or closure by secondary intention. Systemic antibiotics are not usually required for the usual superficial wound infection.

While it is impossible to sterilize the contents of the bowel completely, most surgeons attempt to decrease the population of the colonic bacteria by some preoperative antibiotic preparation.[34]

Such a preparation *theoretically* will decrease the likelihood of postoperative infections, but its injudicious use will result in the proliferation of resistant strains and increase in the likelihood of catastrophic septic complications and the development of pseudomembranous enterocolitis.[4] Washington et al.[48] advocate a combination of neomycin and tetracycline as a preoperative bowel preparation and condemn the reliance upon aminoglycosides as a prophylaxis because of the predisposition to postoperative anaerobic infections. Stone[44] and Polk[36] have demonstrated the efficacy of parenteral prophylactic antibiotic administration in situations wherein the likelihood of infection is great or the consequences of such infection are catastrophic. In "clean-contaminated" (most operations upon the gut) and frankly contaminated cases, we utilize cephalozin intravenously 12 hours prior to operation and continue its administration for a total of three doses in the preoperative period.

The frankly obstructed or grossly diseased colon cannot be cleansed nor adequately prepared. In these situations, resections may be done with safety, but primary anastomosis should be avoided. Here the prudent surgeon will consider the wound to be contaminated and supplement his technique by delayed closure.

Wound Separation

Improper closure and inadequate healing of the abdominal wound may result in postoperative complications ranging from minor separations to complete dehiscence and evisceration.

Most major wound separations are associated with the use of some

variety of a longitudinal incision inadequately supported by retention sutures. The use of the transverse incision is perhaps the single most important factor in the prevention of these complications. The transverse incision divides the transversalis fascia in the direction of its fibers parallel to the vectors of stress. Further, there is minimal denervation and devascularization of the abdominal wall musculature.

When separation has occurred, any exposed bowel should be covered with moist, sterile dressings, the abdomen supported, and the patient immediately removed to the operating room thereafter for definitive closure.

Debridement of the wound is then accomplished, any occult abscesses drained, and the abdominal cavity copiously irrigated with from 10 to 20 liters of saline solution. Some surgeons advocate the use of antibiotic irrigants, but these may offer no advantage to saline in the prevention of postoperative sepsis.[37]

Reclosure is effected with a stout nonabsorbable suture. In longitudinal wounds, a through-and-through suture well back from the wound margin is necessary. Their necrotic effect on the abdominal wall is minimized by the use of rubber tubing or plastic struts. For transverse wounds, we employ a single layer of No. 4 monofilament stainless steel wire. The suture should include all layers except the skin, and the wound is superficially left open to heal by secondary intention.

On occasion, the extent of separation may be too great to permit reapproximation of the edges. In these situations, it may be judicious to employ a Marlex mesh to fill the defect.[32] Aggressive measures to prevent further peritonitis are undertaken including intestinal decompression, diversion of the fecal stream, when appropriate, and massive antibiotic administration. Parenteral hyperalimentation is useful to restore these patients to the anabolic state.

Incisional Hernia

A late wound complication may be the development of an incisional or ventral hernia. The incidence following operation for inflammatory bowel disease is from 0.5[41] to 3.9 per cent.[13] This is no higher than the expected incidence for laparotomies in general.[1] The choice of the incision and the presence of drains or infections all influence the rate of occurrence of this complication. Wound infection is noted in about 25 per cent of patients who develop incisional hernia.

SEPSIS

The most dreaded postoperative complication of intestinal surgery is peritonitis. A certain amount of contamination occurs during every operation, but gross contamination by fecal spillage, inadvertent rupture of an inflamed viscus, or failure of an anastomosis presents an overwhelming challenge to the peritoneal cavity with resultant, and often fatal, bacterial peritonitis.

The biologic relationships of bacteria are of great significance in the

production of peritonitis. There is synergistic action between aerobes and anaerobes, and so-called nonpathogenic bacteria may exist in synergy during fecal peritonitis.[3] In general, the greater the number of bacterial species present in the peritoneal exudate, the more virulent and clinically severe the peritonitis.

The peritoneal lining of the abdominal cavity represents a large surface that approximates the total body surface area. Any trauma to this membrane, either planned or accidental, results in hyperemia and edema. Contaminating bacteria are usually dealt with adequately, but clinical peritonitis will result when the bacterial inoculum is too large or virulent, or if the host's local defenses are compromised because of chronic illness, the presence of intraperitoneal foreign bodies or blood, or drugs that interfere with the inflammatory response. The exudative potential of the peritoneum is responsible for the sequestration of tremendous volumes of fluid during peritonitis. These third space losses may range from 4 to 12 liters during a 24-hour period.[12] This fluid is not utilizable by the patient for circulatory support and must be parenterally replaced. The impaired and inflamed peritoneum will allow the absorption of toxic materials from the peritoneal cavity—particularly bacterial toxins and metabolites.[49]

Totally, the terminal stage of peritonitis is metabolic degeneration from combined fluid volume changes, circulatory insufficiency, disturbed oxygen transfer, and metabolic acidosis.

Postoperative sepsis may appear as either localized or generalized peritonitis. While the diagnosis can be difficult, a surgical infection must be distinguished from a nonsurgical infection. Awareness by the surgeon of the possibility of technical error leading to a localized abscess or, more unfortunately, anastomotic disruption and generalized peritonitis are keys to early diagnosis. An unexplained fever, a pH indicating metabolic acidosis, a tachycardia inappropriate to the clinical situation, the diminution of bowel sounds and, finally, the appearance of frank ileus or the accumulation of intraperitoneal gas on roentgenograms of the abdomen point to peritoneal sepsis. In our experience, classic leukocytosis or abdominal pain is not of great value and their absence can be misleading, particularly in patients receiving large amounts of corticosteroids. Abdominal wall rigidity is, however, a reliable indication of peritoneal irritation.

Once the diagnosis of peritoneal sepsis is established, the surgeon must then differentiate between localized and general peritonitis and provide an appropriate operative attack, adequate chemotherapy, and metabolic support.

TREATMENT OF GENERALIZED PERITONITIS

Antimicrobial Treatment

The rational use of antibacterial drugs should be based upon two principles. The first is to establish a bacteriologic diagnosis and the second is to make an accurate estimate as to the chemotherapeutic agent

that will be effective in vivo. From a practical standpoint, these objectives may not be attained in each patient prior to the institution of treatment. When dealing with infection of enteric origin, the clinician may assume that the offending agents are also enteric in origin, are both aerobic and anaerobic, and are usually a mixed or multiple infection.[3] The positive blood, wound, or peritoneal cultures may be lacking at the crescendo of the patient's illness. Further, imperfect collection and culture techniques may fail to reveal anaerobic organisms that are abundantly present.

Most clinically important anaerobes are susceptible to penicillin with the exception of the most common organism in the colon, Bacteroides fragilis, which is penicillin resistant.[25] Chloramphenicol and clindamycin are effective against this and most other anaerobic species.[25]

For the treatment of postoperative peritonitis, we advocate a combination of drugs. Gentamycin or kanamycin may be administered in combination with clindamycin or chloramphenicol. Our preference is gentamycin, 3 to 5 mg per kg per 24 hours intravenously or intramuscularly and clindamycin, 750 mg intravenously every 8 hours. These combinations will effectively treat both aerobic and anaerobic infections.

If clostridial organisms are identified, or if the clinical situation suggests the possibility of tetanus infection, both active and passive immunity are supplied to the patient.[8]

Operative Treatment of Acute Peritonitis

Although antibiotics are essential in the treatment of peritonitis, the decision to treat peritonitis by operation is usually determined by identification of the source of the contamination. Spreading peritonitis in the postoperative period indicates an intra-abdominal calamity that will require operative correction. Primary repair of the contaminating source is usually impossible or inadvisable; the surgeon will do well to rely on the principle of "diversion of the fecal stream." These operative maneuvers are, of course, supplemented by both antimicrobial and supportive measures.

Occasionally, the source of peritoneal contamination is a sequela of the original disease state and operation, but not a direct consequence of the technical maneuvers of the operation. Such instances include free perforation of a peptic ulcer, strangulation obstruction of the small bowel, ischemic necrosis of a hollow viscus, etc. In such circumstances, the complication is treated on its own merits by closure or resection.

There are, however, certain catastrophes that occur in the postoperative period in the patient who has undergone operation for inflammatory disease of the bowel that require specific operative correction by an experienced surgeon.

Retained Rectal or Sigmoid Stump. A common cause of peritonitis is the retained rectal or sigmoid stump. For a number of reasons, the prudent surgeon may wish to perform total colectomy via a two-stage procedure, or may wish to preserve the rectosigmoid for possible future anastomosis with the small bowel. In these instances, it is far safer to

establish a mucous fistula from the retained distal segment at the cutaneous level. A slight modification of this technique is to close the distal stump and to exteriorize the closed end at the fascial level. If closure is successful, the patient is not bothered by the mucous stoma. If closure is unsuccessful, leakage will occur to the exterior.

A third, but less reliable, option is to resect the rectum beneath the peritoneal reflection and to afford an extraperitoneal closure. The inexperienced surgeon may attempt closure of an overtly or occultly diseased segment of rectosigmoid and replace it into the peritoneal cavity. This is fraught with dangers as the closure may disrupt, leading to generalized peritonitis and bowel obstruction.

Should one be faced with this problem, it may be handled by a variety of means. The safest and most reliable is reoperation to exteriorize the peritoneal portion of the rectosigmoid as a mucous fistula. If the segment is too short or too bound down by the inflammatory response, the surgeon may elect intubation of the proximal lumen and attachment to the parietal peritoneum. This will result in a controlled fistula. Occasionally, in minor peritoneal soiling, the rectum may be resected and reclosed beneath the peritoneal reflection. This option is rarely used because of the inherent dangers of pelvic sepsis.

ANASTOMOTIC DISRUPTION. Disruption of an enterocolic anastomosis is a catastrophe that requires early recognition. The first step in its recognition is to accept the definite diagnostic possibility. Too often, the surgeon is loath to admit to himself such a possibility and needlessly procrastinates. If the enterocolic anastomosis is readily accessible within the free peritoneal cavity, the area of the anastomosis may be exteriorized. Such an exteriorization is best accomplished through a separate incision, although the original incision is used to obtain access to the peritoneal cavity. The surgeon may elect to resect or take down the previous anastomosis and convert it into a temporary enterostomy (usually ileostomy) and distal mucous fistula. In these instances, the ileostomy is fashioned with the same care and attention as during a primary operative procedure.

If an ileoproctostomy has been previously performed, so that exteriorization is difficult or impossible, a proximal loop ileostomy supplemented by copious irrigation of the peritoneal cavity is performed. This is a simple procedure that can be performed expeditiously and may yet be life saving. After healing, the ileostomy can be simply repaired and intestinal continuity re-established. This time interval is usually from 4 to 6 weeks.

Irrigation with a sterile saline solution is of use in diluting and washing out contaminating bacteria and debris. After fibrin forms, however, such irrigation is of limited value. Irrigation with antimicrobial solutions is not generally accepted.[10] Postoperative peritoneal irrigation through indwelling intra-abdominal catheters is favored by some authors,[15] but its advantages are not clear-cut and there is an ever-present danger of erosion of the edematous, boggy bowel by the indwelling catheters.

We have recently utilized peritoneal debridement in severe peritonitis in order to remove peritoneal debris, pseudomembranes, and exudates. Although tedious and time consuming, if carefully done, peritoneal debridement can diminish postperitonitis abscess formation and result in increased survival. Debridement is accomplished by removal of membranes from all visceral and parietal surfaces throughout the abdomen supplemented by copious irrigation.[21]

Localized Peritoneal Sepsis

Intraperitoneal abscesses occurring in the postoperative period represent areas of localized infection subsequent to secondary or postoperative peritonitis. Such an abscess is a continuing site of sepsis that has been isolated by the inflammatory response. The bacterial flora of these abscesses are identical to those of generalized peritonitis.

The diagnosis of abscess is made by a number of circumstantial factors including a spiking fever, partial bowel obstruction, profound leukocytosis, pleural effusion, air-fluid levels visible on roentgenograms, etc.

Pelvic Abscess

Depending upon the extent of the operative dissection, a postoperative pelvic abscess may be drained by a number of routes. The surgeon may wish to approach a large pelvic abscess transabdominally, but extraperitoneally. After locating the site of the abscess, appropriate dependent and/or sump drainage is established. Pelvic abscesses may be drained through the vagina in the parous patient. Vaginal drainage in immature females has proven, in our hands, inadequate and dangerous. If the rectum is in place, the pelvic abscess may be drained through the rectum, but this procedure is less applicable to the patient with inflammatory bowel disease than non-colitic patient. If the rectum has been removed or the supralevator space has been dissected, the pelvic abscess may be drained through the perineum. This drainage is usually accomplished in the presacral space and extends above the remnants of the levator sling.

Subphrenic and Subhepatic Abscesses

These collections of pus may be described by their relation to the falciform ligament and the liver. A high index of suspicion plus several diagnostic radiographic studies are an aid to diagnosis. Pleural effusions, displacement of the gastric outline, and air fluid levels within the abscess may all indicate such an abscess on flat or upright chest and abdominal roentgenogram. A transmission-emission scan utilizing cobalt 60 and technitium 99 to determine separation of the pulmonary and hepatic parenchyma have proven extremely helpful to us in establishing the presence of a subphrenic, suprahepatic abscess. Subphrenic abscesses are best drained extraperitoneally. For anterior abscess a Clairmont subcostal incision is advocated, and the posterior approach of Ochsner through the bed of the 12th rib is ideal for posterior presentations.[6] Adjuvant antimicrobial therapy is, of course, utilized.

Retroperitoneal Abscesses

Altemeier and Alexander[2] have described the anatomic boundaries of the retroperitoneal space. Most retroperitoneal abscesses associated with inflammatory bowel disease occupy the anterior retroperitoneal space. Only rarely does enteric disease result in a posterior retroperitoneal infection. The majority of these abscesses are present preoperatively and their appearance in the postoperative period implies that they were overlooked or inadequately drained at the time of operation. Vigorous and thorough drainage and appropriate antimicrobial therapy are essential for proper treatment.

Intramesenteric Abscess

Occasionally, loculations of pus will occur between leafs of mesentary or loops of small bowel. These abscesses are not amenable to extraperitoneal drainage and their treatment may require transperitoneal drainage or bowel resection. Their intimate association with the small gut makes definitive drainage often impossible without resection.

PERSISTENT PERINEAL WOUND OR SINUS

The persistent perineal wound or residual sinus is merely a failure of healing of the posterior wound. An open or infected wound or sinus remaining after six months is usually considered as fulfilling the definition of persistence.

While many of the persistent wounds are merely minor cutaneous sinuses of no practical importance to the patient, others are huge, purulent cavities with unhealthy granulations that interfere with the patient's health, rehabilitation, and social acceptance. The incidence of the unhealed perineal wound following total proctocolectomy is quoted as from 10 per cent[13] to over 40 per cent.[24] Of more practical importance is that prolonged hospitalization is often necessary for the closure of these wounds, and multiple operative procedures are entailed.[39]

Corticosteroids apparently play no role in the persistence of the open wound.[23] Granulomatous colitis is more often associated with nonhealing than is ulcerative colitis.

The development of this late complication is more common in women, particularly when the perineal wound is packed. This suggests that lack of pelvic support plays an etiologic role. An occasional wound failure, when investigated, will reveal a ready explanation for persistence: retained mucosa, fistula to bladder or gut, neoplasia, etc. When these uncommon situations exist, the removal of the cause and re-excision of the walls of the wound will suffice for cure.

The avoidance of pelvic sepsis, combined with primary closure of the perineum and the early removal of small, effective drainage is the best prevention of this complication.

We would add three other observations that aid in the prevention of the persistent perineal wound. First, is to obtain precise hemostasis in the pelvis and levator area and to avoid the introduction of foreign

bodies such as packing. We use only a small posterior sump drain in the presacral space for 24 to 48 hours after operation. Secondly, the perineal wound should be closed in layers, completely closing the levator ani, the subcutaneous tissue, and the skin, without the formation of a dead space. Of paramount importance is to completely excise all of the circumferential voluntary anal sphincter. The surgeon's desire for conservatism and for preservation of the perineum will result in his excising the rectum, while leaving intact the sphincter. Postoperatively, this intact sphincter acts as a deterrent to drainage and will create a dead space. This, in turn, leads to secondary infection and the development of a persistent wound.

If the surgeon is confronted by a large, infected persistent perineal wound he must first investigate the patient to be sure there is no fistula, foreign body, or unrecognized abscess that is to blame. If these are present, they are treated appropriately. Usually, however, he will be faced with a chronic, thick-walled, fibrous cavity bearing pale, infected granulations. Under appropriate anesthesia, the entire thick fibrous wall of this cavity must be excised. Curettement is inadequate treatment because it will usually not remove the thick fibrous wall. During this procedure, the surgeon must be scrupulously careful to avoid injury to the anterior bladder and urethra and must also realize that the small bowel may be in close proximity to the superior portion of this cavity. This wound is usually left open, irrigated frequently with saline, and the patient discharged in a few days. In the overwhelming number of cases, this wound will then heal spontaneously in a few weeks.

If, however, there remains a cutaneous defect that is slow in healing, or if the persistent wound is merely a cutaneous defect without superior projections, healing is aided by a skin graft. We have been gratified by applying a meshed split-thickness skin graft utilizing 12/1000 of an inch graft meshed to a 1:3 ratio. This graft may be applied with a stint or may be left open as suits the circumstance and local contamination.

COMPLICATIONS ASSOCIATED WITH ILEOSTOMY

The problems associated with a permanent ileostomy are of major importance when considering proctocolectomy for inflammatory bowel disease. Jacob, Pace, and Thomford[22] reported that 14 per cent of their patients required revision of the stoma within the first year, and one-half of the patients required revision within 10 years.

Ileostomy Dysfunction

In 1951, Warren and McKittrick[47] defined ileostomy dysfunction as partial obstruction of the stoma resulting in cramps and increased stomal output. The advent of the Brooke ileostomy in 1952[9] substantially eliminated this entity. Nevertheless, stomal partial obstruction is today seen as a result of improper ileostomy construction. The leading causes of stomal dysfunction today are: construction of a short or

ischemic ileostomy with local scarring or retraction; failure of the surgeon to allow an adequate excision of skin, subcutaneous tissue, and fascia at the ileostomy site; and recurrence of stomal or prestomal inflammatory bowel disease. These situations are today prevented or remedied by adequate establishment of an ileostomy and resection of recurrent disease when indicated.

At the time of the construction of the ileostomy, several safeguards may be observed which will effectively prevent postoperative stomal obstruction. First, the site of the stoma is chosen with care so as to avoid scars, fistulae, the umbilicus, bony prominences, etc. This site should be marked by tattooing the skin at the site selected. We prefer either a Brooke or a Goligher extraperitoneal ileostomy and use them routinely. The Goligher ileostomy is not, however, recommended following proctocolectomy for Crohn's colitis. The terminal ileum is brought out through the selected site so that 4 cm of bowel protrudes from the skin margin without tension. The mesentery of the small bowel is always to the right of the patient—this will prevent inadvertent torsion. The stoma site is tailored by excision of a circular area of skin approximating the size of a 10 cent piece. If there is dilated bowel from chronic obstruction, the defect will have to be proportionately larger. A similar amount of subcutaneous fat is removed, care being taken not to undermine the cutaneous defect. The fascial component of the rectus sheath is also excised to approximate the defect in the skin. Failure to excise this fascial layer will routinely result in prestomal stenosis. The abdominal wound is completely closed prior to fashioning the external portion of the ileostomy. The bowel is inverted upon itself by any of a number of techniques. This will allow for a 2-cm protuberance of bowel. A longer ileostomy will often result in pressure necrosis of the bowel on the appliance; a more flush stoma is difficult to manage and the appliances are difficult to maintain in place. The external full-thickness layer of bowel is approximated to the skin by absorbable suture material.

If the ileostomy requires revision, a formal laparotomy is usually necessary to produce a satisfactory result. Rarely, a purely external operation may suffice if the problem is limited to the mucosa of the bowel or cutaneous cicatrix. Less superficial problems are rarely adequately remedied by less than laparotomy, take down of the imperfect ileostomy, and construction of a proper stoma.

Ileostomy Prolapse

Today, this is a rare complication of ileostomy. Prolapse may be uniformly prevented by the secure anchoring of the mesentery of the bowel to the transverse layer of the abdominal wall by nonabsorbable suture material adjacent to the internal abdominal wall defect. Sutures which include the serosal surface of the bowel should be avoided as these do not prevent the sliding hernia and may cause fistulae.

Paraileostomy Hernia

A prolapsed ileostomy may present as an interstitial hernia with protrusion of ileal tissue into the subcutaneous layers of the abdominal

wall. A paraileostomy hernia is diagnosed by the presence of obstructive symptoms, local pain, and the palpation of the hernial contents. Revision of the ileostomy with excision of the sliding sac and lateral repair of the abdominal wall defect is the treatment of choice.

Ileostomy Fistulae

Ileocutaneous fistulae are a relatively common problem that may necessitate ileostomy revision and/or relocation. Fistulae are caused by: (1) damage to the ileum at the time of operation; (2) ill-fitting appliance and mechanical irritation of the ileum; (3) self-manipulation of the stoma; (4) chronic and continued compromise of the ileostomy by such entities as a tight fascial ring; and (5) the occurrence of stomal or prestomal ileal inflammatory disease.

If the ileostomy fistula occurs above the level of the skin, the output will be diverted from the orifice and will not cause serious local sepsis. If, however, the fistula is at skin level or below, abscess and wound infection invariably result. This will necessitate drainage of the abscess and fistula and revision of the ileostomy. If the right lower abdominal wall is massively involved by an inflammatory process, relocation of the ileostomy to another site may be necessary. The decision to relocate should be avoided, if possible, as left-sided ileostomies are notoriously troublesome because of inadequate length and the inability to close the lateral space.

Dermatitis

Selection of the site of the ileostomy, proper protection of the skin, and the fitting of an appropriate appliance will all routinely protect the patient from paraileostomy dermatitis. For detailed accounts of methods of mechanical skin protection by adherents or protective devices, the reader is referred to the numerous medical articles on the subject,[20] as well as bulletins and manuals of the United Ostomy Association.[30]

INTESTINAL OBSTRUCTION

Any abdominal operation may be complicated by intestinal obstruction. The risk is increased after operations for inflammatory bowel disease, especially after proctocolectomy and ileostomy. Complete small bowel obstruction occurred in 7.7 per cent of the patients undergoing operation at St. Mark's Hospital between 1960 and 1964.[41] Obstruction as a cause of late death was second only to carcinoma in the series reported by Ritchie.[40]

In the immediate postoperative period some degree of paralytic ileus is always present. Overly zealous attempts at feeding may result in a prolonged or recurrent ileus. These episodes are readily treated by gastric decompression and cessation of oral feeding. Similarly, incomplete mechanical obstruction appearing in the immediate postoperative period will usually resolve by continued intestinal decompression. Per-

sistent high-grade partial mechanical obstruction will require operative intervention with appropriate lysis of adhesions or resection.

The bowel obstruction associated with recurrence of granulomatous disease at or near a site of enterocolic anastomosis is usually partial, remitting, but progressive. This is a classic complication of the disease process and is an indication for reoperation. deDombal[13] comprehensively studied the results of surgical treatment of Crohn's disease and concluded that operations short of colectomy were subject to reoperation of some type in over one-third of patients. Obstruction was the second most common complication necessitating reoperation in his series.

Complete mechanical small bowel obstruction in the postoperative patient is an urgent indication for operation. Ellison[18] observed an improved survival and a lower rate of complications after prolonged intestinal intubation was abandoned in favor of rapid preparation and early operation. By following this program, the incidence of intestinal gangrene was only 10 per cent and the overall mortality rate was 4 per cent.

Mechanical obstruction in the inflammatory bowel disease patient may be on a nonspecific basis from adhesions. However, there are three potential sites of hazard particularly prominent in these patients: lateral space hernias with incarceration at the site of the ileostomy or colostomy; adhesions to the pelvic floor; and herniation through the pelvic floor with entrapment. The treatment of these situations is facilitated by early recognition followed by appropriate operation. The prevention of these serious complications may be aided by the secure closure of the lateral spaces. The parietal peritoneum is secured to the cut edge of the mesentery rather than to serosa of a potentially diseased viscus.

The pelvic floor may be secured by a number of methods. In order to prevent postoperative adhesions, a closure under tension should be avoided. A tense closure is also prone to dehiscence and entrapment of bowel. If the pelvic peritoneum remaining at the termination of the procedure is inadequate for easy reapproximation, the surgeon should either leave the pelvis widely open or, if the perineal wound is also inadequate for closure, achieve support by packing the perineum and pelvis over a protecting sheet of Owens surgical material. The Owens material is quite nonreactive and satisfactory regrowth of the peritoneum can be anticipated in 1 week's time.

MALABSORPTION–SHORT BOWEL SYNDROME

Serial, massive, or injudicious resections of the small bowel, particularly for patients suffering from Crohn's disease, may result in the complexities of intestinal malabsorption known as the "short bowel syndrome." The loss of adequate small bowel function results in protracted diarrhea and the malabsorption of essential nutrient substances. Resection of from one-third to one-half of this may represent the upper limit of safety. When resections exceed this – particularly in the absence

of the colon—absorption is markedly altered and poses significant management problems. In addition, the loss of a specialized function of the ileum can lead to vexing diarrhea and specific malabsorption even after seemingly "conservative" resections.

Inadequate absorptive area and short transit time leads to incomplete digestion of carbohydrates, fats, and proteins, and efficient segmental absorption cannot occur. Loss of ileal function leads directly to malabsorption of bile salts and vitamin B-12. Subsequent diminution of the enterohepatic circulation of bile acids gives rise to adequate micellar formation and contributes to malabsorption in the remaining jejunum. Consequently, severe steatorrhea is more prominent after ileal resections than jejunal loss. The presence of fatty acids and bile acids in the colon seems to interfere with salt and water absorption, further promoting diarrhea.[5] This so-called "bile salt enteropathy" is often successfully treated by oral cholestyramine, 8 to 12 gm per day. This preparation binds unabsorbed bile acids to prevent their effect upon the colon. Loss of vitamin B-12 will result in a megaloblastic anemia and combined system diseases. Steatorrhea and fat malabsorption causes loss of the fat-soluble vitamins A, C, and K. Deficiencies in vitamin D and increased excretion of calcium and magnesium with fecal fat can lead to severe deficits in these minerals and serious neurologic and metabolic abnormalities.[45]

Therapy for patients with short bowel is directed at the replacement of fluid and electrolytes, adequate caloric intake, and supplementation of inadequately absorbed nutrients, vitamins, and minerals. Some degree of eventual intestinal adaptation can be expected and the malabsorption should improve. The capacity of the ileum to adapt is greater than that of the jejunum.[16] Initially, we rely on total parenteral hyperalimentation during the immediate postoperative period during the institution of oral feedings. Hyperalimentation can reverse the catabolic effects of operation and nutritionally support the patient while intestinal anastomoses are healing and the bowel is starting to adapt.

With the resolution of postoperative ileus, the remaining bowel's absorptive capacity can be more accurately assessed and oral intake resumed. Treatment at this point is based on overcoming specific absorptive defects so that the patient may be weaned from hyperalimentation. Intestinal adaptation takes place over a variable period of time and may require months. The transition to oral feedings must be gradual. At first, feedings must be small and frequent. We utilize an elemental diet similar to that reported by Voidk, et al.[46] Such diets demand no digestive effort by the compromised gut and need a minimal absorptive surface. Proteins are given as hydrolysates or amino acids, and fats are delivered as medium-chain triglycerides which are easily absorbed directly into the portal circulation, contributing minimally to steatorrhea. We limit the intake of the triglycerides to about 20 gm per day. The major drawback of these diets is their unpalatability. Usually, they must be infused via a soft nasogastric feeding tube. In addition, these formulas are uniformly hyperosmolar.

UROGENITAL COMPLICATIONS

Problems affecting the urogenital system in the postoperative period are either a direct result of operative trauma or residual inflammatory disease, or indrect sequelae of metabolic derangements coincident to ileostomy dysfunction.

By far, urinary and sexual problems occur most frequently after resection of the rectum.[7] One or both ureters may be injured, perineal-urinary fistulas may develop, urinary retention may be demonstrated, and the male patient may become impotent. Basically, all of these complications are associated with operative trauma or manipulation.

If ureteral injury has been sustained, diagnosis will usually be accomplished by excretory or retrograde pyelography. The level and the type of injury will dictate the appropriate therapy. Most ureteral injuries occur at the upper level of the rectum immediately below the peritoneal reflection. In most instances, ureteroneocystostomy can eventually be established after adequate drainage or diversion of the urinary stream has allowed inflammation to subside. Occasionally, nephroureterectomy is necessary.

Bladder or urethral fistulae to the perineal wound will usually close spontaneously with long-term ureteral catheter drainage. Occasionally, secondary closure supplemented by cystostomy may be warranted.

Persistent right-sided hydroureter occurring in Crohn's disease has been reported.[19] Ureterolysis of the involved ureter and appropriate drainge of the associated retroperitonitis will uniformly lead to recovery.

Postoperative Bladder Dysfunction

Difficulty in bladder emptying has been reported following total proctocolectomy in up to 16 per cent of patients.[7] Both male and female patients may suffer this complication. If prolonged urethral drainage fails to result in spontaneous voiding, the patient should be investigated by excretory pyelography and cystometrogram. Partial or complete motor neuron paralysis is the usual cause of the dysfunction. In these patients, the pelvic autonomic nerves have probably been damaged at the sacral promontory or lateral and anterior to the rectum in the retrovesicle and retrouterine spaces. To avoid this problem, dissection should be carried out close to the rectum and medial to the endopelvic fascia. Decompensated or atonic bladders of either sex are often rehabilitated by a program of cholinergic stimulation.

If endoscopy reveals vesicle neck contracture or obstructing prostatic hypertrophy, correction of these lesions is best performed several weeks after coloproctectomy. Transurethral resection of the offending obstruction is the procedure of choice. This condition is usually seen in male patients, but occasionally females are similarly affected by urethral stricture.

Sexual Dysfunction

Although sexual dysfunction following adequate abdominoperineal resection for cancer may be considered as a necessary consequence of

an adequate cancer operation, it is distressing and unnecessary to render the young male with inflammatory bowel disease impotent.[33] Sexual function in the male is under the control of the autonomic pelvic plexus and avoidance of injury to these delicate structures is the only preventative for postoperative sexual dysfunction. These nerves can be avoided by dividing the mesentery as close to the bowel as possible at the sigmoid colon and rectum and by preserving a generous portion of pelvic peritoneum.[28]

If impotence is persistent after 3 months, palliation may be afforded the young male by the operative insertion of a flexible Silastic prosthesis into the shaft of the penis.[35]

Ureteronephrolithiasis

From 7 to 13 per cent of patients with ileostomies eventually develop urinary stones. Deren found calcium stones, uric acid stones, as well as "unclassified" stones. Previous steroid therapy, urinary tract infection, hyperuricemia, hypokalemic alkalosis, consistently acid urines, etc. have all been implicated in the genesis of these stones.[14]

Excessive ileostomy loss with concomitant decreased urinary volume appears to be an attractive thesis for the genesis of these stones.[31] Also implicated in the occasional patient is excessive bile salt loss and the associated derangement of the calcium metabolism.

Maintenance of an adequate urinary output, correction of electrolyte depletion, alkalinization of urine, treatment of infection and the use of xanthine inhibitors in patients with hyperuricemia are the appropriate measures for the prevention of recurrent stone disease.

NEUROLOGIC COMPLICATIONS

The central nervous system complications associated with the administration of steroids, septicemia and shock, may occasionally be observed in the patient suffering inflammatory bowel disease. These complications are, however, not specific to inflammatory bowel disease. Postoperative seizures are occasionally seen, particularly in young patients suffering inflammatory bowel disease. Steroid administration, fluid retention, and arterial hypertension are common to these patients.[26]

Peripheral neuropathies involving the branches of the sciatic nerve are frequently observed in varying severity in those patients who have undergone total proctocolectomy in the lithotomy position. These neuropathies are a direct result of pressure or trauma to the peripheral nerves coincident to the position of the patient.[26]

HEPATITIS

Liver disease is a common complication in patients with ulcerative colitis.[17] There is a histologic liver abnormality in the overwhelming number of patients with inflammatory bowel disease: 70 per cent show

inflammatory changes and 3 to 5 per cent of the patients have cirrhosis. Stokes has observed that patients with pre-existing enteritis are more subject to attacks of hepatitis during an epidemic than noncolitics.[43] While the majority of the liver disease observed in patients with inflammatory bowel disease is probably of portal, bacterial origin, there appears to be more than a coincidental occurrence of viral hepatitis in patients with inflammatory bowel disease.

Pyogenic abscesses of the liver are rare in uncomplicated inflammatory bowel disease. Their appearance is usually associated with a complication of operation such as severe peritonitis.[27]

CONCLUSION

Operations for inflammatory bowel disease are attended by specific complications that may be serious or fatal. A knowledge of the hazards involved in the operative treatment of these patients will, hopefully, contribute to a lessening of the complications and deaths and improve the ability of the surgeon to safely treat his patient.

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Combined Abdominoperineal Resection

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Combined abdominoperineal resection is the treatment of choice for rectal cancer as well as certain inflammatory large bowel diseases. This surgical procedure is performed both for cure and palliative control. A rectal neoplasm should be removed whenever possible because such cancer, even with metastases, seems to have an increased survival rate when resected.[2, 17] Rectal excision can also improve the quality of survival, alleviating bleeding, severe tenesmus, gluteal pain, and foul-smelling anorectal discharge.

This approach to eradicate the neoplasm or palliate its symptomatology may be followed by any of the complications of major abdominal surgery, including pulmonary embolus, respiratory distress, myocardial infarction, cerebral vascular accident, hypovolemic shock, intestinal obstruction, and paralytic ileus.[8] This article will deal only with complications that are more specific for abdominoperineal resection. An attempt will be made to categorize the problems into immediate, early, and late complications.

Morgan[14] postulates that the habitus as well as sex of the patient may influence the incidence of complications after abdominoperineal resection. The most suitable patient is the slender female with a wide pelvis. Unfortunately, the habitus of the patient is beyond the control of the surgeon, and the urgency of the surgery precludes any attempts at preoperative weight reduction.

HEMORRHAGE

During the surgical removal of the rectum from its deep pelvic location, the most common cause of massive bleeding is the destruction of the presacral venous plexus. This plexus is well protected by the presacral fascia, but during sharp dissection of the posterior rectum from either the abdominal or the perineal approach, the deep presacral space

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may be entered. In view of this, the surgeon should bluntly dissect the entire rectum from the presacral fascia from the abdominal approach. If perchance the presacral venous plexus is damaged, control is often aided by the use of steep Trendelenburg position and adequate compression. Hemostasis is best achieved by careful suture ligation, whereas cautery is seldom effective and packing is useful only for the period of time that the rectum is retained. If pelvic hemorrhage cannot be controlled by the aforementioned techniques, bilateral internal iliac artery ligations may prove helpful.[1]

Once the immediate hemorrhage has been controlled and the area of the presacral plexus appears dry, the surgeon must still keep in mind that early postoperative hemorrhage is now more likely to occur than if the plexus had never been damaged. A posterior perineal pack is used during the first 72 hours to ensure continued postoperative hemostasis. The pack is a 5-yard soft cotton gauze treated with 30 to 40 ml of Peruvian balsam, which controls odor and stimulates granulation tissue. The pack is removed after 72 hours and thrice daily irrigation with 1 per cent neomycin/Ringer's lactate solution is initiated.

To close the perineal wound over suction drains may endanger the precarious hemostasis and lead to massive early or late hemorrhage.[8]

Late hemorrhage, 6 to 10 days after operation, is a very rare occurrence and is associated with infected granulation tissue in the perineal cavity. To minimize the occurrence of late hemorrhage, Malt and Nundy[13] suggest that the peritoneal floor of the pelvis not be reconstructed. Several other investigators[15, 16] recommend this approach, which allows small bowel to fill the deep perineal dead space.

If the surgeon elects to close the perineal skin tightly over suction drains, and late hemorrhage occurs, he must return the patient to the surgical suite for perineal exploration, curettage of the infected granulation, and open packing of the wound.

As one might expect, the incidence of late hemorrhage of the perineal wound is more common in patients who had inflammatory bowel disease with rectocutaneous fistulae. In spite of prophylactic antibiotics, sepsis and subsequent hemorrhage still occur, and this may be related in part to the cortisone therapy these patients receive.[22]

URINARY TRACT INJURY

Urinary tract injury may be deliberate or unrecognized. The deliberate injury such as en bloc resection of ureter with rectal excision usually does not progress to a complication. It is the accidental, unrecognized injury that eventually produces sequelae that must be dealt with. These injuries are immediate in nature, but are usually not recognized until the early or late postoperative period. An early presentation of this problem is the appearance of progressive, severe flank pain with oliguria. These symptoms should alert the surgeon to the possibility of ureteral ligation. The acute distention of the proximal collecting system initiates a pain similar to acute pyelonephritis. To document the injury,

a postoperative intravenous pyelogram should be done, and if contraindicated because of allergy to the dye, cystoscopy with retrograde pyelography should be performed. Another technique to assess the possibility of ureteral damage is to instill methylene blue dye intravenously during cystoscopy. If the ureters are patent, the observer will visualize blue stained urine from both ureteral orifices.

The early complication of abdominal distention, oliguria, and shifting dullness on percussion suggests the possibility of complete ureteral transection or bladder dome disruption. Again, cystoscopy and retrograde cystourethrogram must be done immediately to assure an intact bladder mucosa and patency of the ureteral orifices. The ureters are most vulnerable to injury at the site of the pelvic brim and at their insertion into the bladder.[1, 8] To prevent severance or ligation of the ureters, these structures must be well isolated and visualized when the mesenteric vessels are ligated and the lateral ligaments are clamped and ligated. Blind clamping of lateral stalks with crushing clamps to control hemorrhage is a technique that enhances the chance for ureteral injury.[1]

To repair a simple total or partial severance of a ureter, the situation is best handled as if it were a vascular repair. An end-to-end anastomosis using fine, polyglycolic acid (Dexon) suture is best. The patency and proximal decompression of the ureter is assured by using an adequate size ureteral catheter as a stent.[8]

If there is an actual loss of a ureteral segment, and an end-to-end anastomosis is impossible, a cross ureteroureterostomy has been suggested.[12] The technique, however, may jeopardize the uninjured side. In some cases, the safest and most expedient way to deal with a significant ureteral tissue loss is to perform a nephrectomy, provided the opposite kidney is known to be functionally and anatomically normal.

Some surgeons[8, 18] would rather ligate the proximal damaged ureter, thus hoping to produce atrophy of the corresponding kidney. This ligated kidney may produce silent atrophy, but often this does not occur and hydronephrosis with severe pain or infection results.

When both ureters are damaged with marked tissue loss and primary anastomosis is technically impossible, an ileal conduit may be fashioned.[4]

Bladder injury is usually a simple laceration of the fundus of the bladder. This iatrogenic complication usually occurs with careless entry into the abdomen. A simple two-layer closure with absorbable suture and adequate bladder drainage is the treatment of choice.

The bladder complication faced by most surgeons is direct extension of the tumor into the posterior vesical wall. There are only two alternatives which are feasible: partial or total cystectomy. Total pelvic exenteration with ileal conduit and terminal sigmoid colostomy is the treatment of choice if the patient is a candidate for surgical cure. If the combined abdominoperineal resection is for palliation, a partial cystectomy should be considered. If the partial cystectomy involves the trigone of the bladder, the surgeon must reimplant the ureters into the bladder. When the partial cystectomy is extensive and reimplantation of the ureters is impossible, an internal ileal conduit may be constructed.[8]

It is our opinion that not all patients with bladder involvement are candidates for extensive surgery. The surgeon must consider the quality of life that the patient faces in the future. He must consider the question, is this tumor truly operable and is this surgery beneficial to the patient? The answer to this question becomes yes if the pelvic organs are not fixed with tumor tissue to the pelvic wall, such as the frozen pelvis, and if no distant metastases are present. After extensive partial cystectomy most patients develop urinary frequency which may last throughout their life expectancy. This frequency may actually be detrimental and produce more complications, thus deteriorating the quality of life.

Prostatic urethral injury commonly occurs during the perineal resection of a large bulky fixed neoplasm. If the prostatic urethra is completely severed, a fine interrupted Dexon suture repair should be attempted with a Foley catheter in place. This injury can be minimized if an indwelling Foley catheter is placed into the bladder prior to the perineal resection. Once the rectum has been removed, the repair of the prostatic urethra may be quite difficult because of lack of suitable tissue for repair, but one may use prostatic tissue itself in a longitudinal tear of the urethra.[8]

It is mandatory that all prostatic urethral repairs be stented by an indwelling urethral catheter. This catheter should be left in place for approximately 14 days. To ensure adequate urinary drainage, a suprapubic tube cystostomy is mandatory.

An important principle with regard to all repairs within the perineal wound is the use of an absorbable suture material such as Dexon. If nonabsorbable suture material is used for hemostasis or urethral repair, it may well be accompanied by stitch abscess and foreign body reaction and eventually lead to persistent draining urethral fistulae.

PERINEAL INFECTIONS

The perineal wound infections are of two types: a diffuse hypertrophic discolored granulation tissue which weeps purulent serum, and the well developed perineal abscess.

Curettage and debridement of this granulation tissue are necessary so as to prevent further late complications. Even if the perineal wound is treated by the open method, some degree of infection occurs in most cases. The infection is readily diagnosed, however, because the open perineum may be approached directly via both digital and proctoscopic examination.[8]

Abscess formation is a late manifestation of infection, and can lead to a myriad of problems including wound evisceration, stenosis of the cutaneous defect and persistent perineal tracts. The utilization of neomycin irrigation in the open technique helps to minimize the chances of abscess formation.

It has been inferred that pelvic abscess formation is more frequent when the perineal defect is treated by tightly suturing the skin over suction drains.[8] This may be due to the hematoma remaining in the dead

space created by improper placement of drains or inadequate drainage. Use of sump drains with a catheter placed for constant irrigation can help prevent abscess formation when using the closed technique. Should perineal tenderness and pyrexia develop following primary perineal closure, the wound must be explored and if infected, opened and treated by the open technique.

When utilizing the open technique, the neomycin irrigations are halted upon discharge and soap suds irrigations performed at home. After each irrigation, the patient places a gauze wick into the wound to aid in drainage and lysis of loculations. If the patient fails in meticulous wound cleansing, a cutaneous stenosis may form.

PERSISTENT PERINEAL TRACTS

The cutaneous stenosis does not allow adequate drainage from the pelvic dead space left behind, and if not removed surgically will most certainly form a persistent perineal tract. Once formed, eradication of the trait becomes a very difficult matter. Multiple curettage with silver nitrate will occasionally seal a persistent tract, but if the tract is completely epithelialized, the entire tract must be removed surgically. When the tract is deep and its origin appears near the pelvic reflection on sinogram, it becomes mandatory to remove the tract by a combined abdominoperineal approach. If a simple perineal approach is used, injury to displaced pelvic organs cannot be adequately ascertained.

Other causes for persistent tracts include retention of packing material or recurrence of the neoplasm. Biopsy of all persistent tracts is therefore mandatory.

POSTOPERATIVE PELVIC PAIN

Postoperative pelvic pain is a late complication of rectal excision. This pain may be intermittent or constantly progressive. Recurrent pelvic tumor can not only cause a persistent tract, but also produce unrelenting persistent gluteal pain and scar tenderness. To minimize this complication preoperative pelvic supravoltage radiation has decreased the incidence of local recurrence of adenocarcinoma of the rectum and rectosigmoid.[20] Persistent and progressive pain following abdominoperineal resection is highly suggestive of local recurrence, and is often the first symptom of such recurrence. The pain has been described as a tightening sensation of the perineal scar, tenesmus, or a stabbing sensation radiating to the testes. One of our patients described a pain similar to dysuria, and urologic examination was normal. Serial elevations of the CEA and perineal biopsy, however, confirmed recurrence of squamous cell carcinoma of the anus. Goligher[8] states that even if recurrence cannot be documented by biopsy or bone destruction on pelvic roentgenogram, the tender area should be treated with super voltage radiotherapy. Surgical exploration of a tender scar is of little benefit, and

there is a high incidence of pelvic organ damage following re-exploration after previous abdominoperineal resection.

A condition which must be differentiated from the painful tumor recurrence is the phenomenon of the phantom rectum. Several authors[5, 6, 8] mention this problem with incidence ranging from 10 to 68 per cent. The cause of this problem is poorly understood. However, this condition produces a sensation of pressure as if the patient must defecate.

Phantom rectum may be only transitory following abdominoperineal resection, but has been known to last as long as 14 years.[8] Our experience revealed an incidence of 23 per cent and all cases were of a transitory nature. An interesting observation in our series was that only those patients undergoing primary perineal closure developed this complication.

There is no specific treatment for this condition other than reassuring the patient that he does not harbor an occult recurrent neoplasm. Serial CEA levels can not only help to alleviate the patient's fears, but also aid the surgeon in documenting true neoplastic recurrence.

SEXUAL DYSFUNCTION

Amongst the late sequelae following abdominoperineal resection in the male is sexual dysfunction. The problem ranges from incomplete erection to complete impotence.

Objective measurement of sexual dysfunction after abdominoperineal resection is a difficult task, as many of these patients have waning sexual function preoperatively, and any minimal autonomic nerve injury may be sufficient to interfere with, or completely abolish sexual function.

Impotence following abdominoperineal resection is presumably due to parasympathetic nerve injury, most likely secondary to avulsion of the nervi erigentes from their origin. This can occur if the presacral fascia has been entered. Impotence can also occur after low anterior resection where the presacral fascia remains intact, and this is most likely due to sympathetic nerve destruction.[8]

Ejaculatory dysfunction occurs with damage to the pelvic sympathetic nerves. Learmonth[11] demonstrated the control of both seminal vesicular contraction and bladder sphincter function by sympathetic nerve fibers. Ejaculation is absent when these fibers are destroyed. Some patients are still able to have orgasm without ejaculation, and this points toward the interaction of neurologic and psychological factors in mediating sexual response.

To minimize parasympathetic and sympathetic nerve damage during pelvic dissection of the rectal tumor, the areolar tissue around the great pelvic vessels should be left undisturbed. Preservation of the areolar tissue from the sacral promontory to the posterior wall of the rectum protects both sexual and urinary functions.

UROLOGIC COMPLICATIONS

Complications related to the urinary system are most common in any deep pelvic manipulation. Such complications consist of urinary retention, incontinence, oliguria, anuria, dysuria, and hematuria. After rectal excision, 50 per cent of male patients demonstrate some degree of adynamic bladder paralysis after removal of the indwelling catheter.[21] The actual cause for postoperative urinary retention is not clearly understood. It is believed that several factors contribute to the problem: prostatic enlargement, chronic urinary infection, loss of pelvic support, and nerve injury.[3, 8-10]

Prostatism is common in the male patient undergoing abdominoperineal resection, because the peak incidence of prostatism correlates well with the peak incidence of rectal cancer. Watson[21] studied 50 consecutive males with urinary retention after abdominoperineal resection and concluded that prostatism was the major problem. He also states that the majority of these patients eventually recovered normal micturition with conservative therapy.

Occasionally prostatic enlargement is difficult to diagnose preoperatively due to the bulkiness of the tumor. Even if the prostate is palpable and felt to be normal prior to surgery, the patient may have a median lobe enlargement causing urinary retention.

The close relationship of the bladder, prostate, and distal portions of the ureters to the rectum make it impossible to preserve the normal supportive tissue to these structures during rectal excision. The large posterior dead space that remains after rectal extirpation and the destruction of the pelvic diaphragm leads to posterior displacement of the bladder, and is further exaggerated with the patient lying in bed. The sharp posterior angulation of both the membranous urethra and the trigone of the bladder accentuate urinary retention with the patient supine. As the patient becomes ambulatory, the angulation is obliterated and micturition becomes normal.[8, 10]

To minimize the importance of this angulation, an indwelling catheter is kept in place for 1 week. The angulation is also diminished when the perineal wound is packed, as with the open technique. The pack acts as a false pelvic diaphragm in support of both the prostate and the bladder. A perineal pack may be retained for 3 days without ill effects and during this time, early fibrinous adhesions help stabilize the distal urinary tract while ambulation of the patient lifts the bladder anteriorly.

The condition of prolonged urinary retention may last as long as 6 to 8 months, and when this phenomenon prevails it is assumed that nerve injury has occurred.

NERVE INJURY

Experimental evidence[11] and clinical observations[3, 8, 9] correlate the destruction of the pelvic sympathetic nerves with increased stimulation

of the bladder neck and internal sphincter as well as inhibition of detrusor muscle function. This is probably secondary to the unopposed parasympathetic function once the sympathetic influence has been removed. With complete division of the pelvic sympathetic system, the patient develops frequent micturition and incontinence.

With deliberate parasympathetic nerve injury, there is contraction of the detrusor muscle, relaxation of the bladder neck, and finally, bladder distention.[3, 9] When this occurs, the syndrome of neurogenic bladder is evident. If this condition is left untreated, overflow incontinence occurs and micturition is accomplished only by straining and the residual urine becomes infected. The residual urine may decrease to zero after several months, but the patient remains incontinent at night.

Parasympathetic nerve injury occurs with radical pelvic dissection and wide excision of both lateral ligaments, as well as bilateral internal iliac lymph node dissection. The nerves which must be preserved to ensure normal micturition are the three nervi erigentes from the second, third, and fourth sacral nerves bilaterally.[8, 9, 11, 21] The destruction of the presacral fascia due to a faulty plane of dissection during the perineal operation can avulse the nervi erigentes close to their origin from the sacral nerves.

The management of urinary retention is of vital importance in the rehabilitation of the patient following extensive pelvic surgery as the condition predisposes the patient to urinary infection. To minimize the incidence of this complication and treat it adequately, an indwelling Foley catheter should remain to closed gravity drainage for a period of 1 week. If the catheter is removed prematurely, the patient will fail to micturate adequately and the catheter must be reinserted, thereby increasing the risk of urethral trauma and infection. Leaving the catheter in place for 1 week will not only avoid this problem, but will also act as a stent for the angulated membranous urethra while sufficient support is being produced by intrapelvic adhesions.

Once the patient is ambulatory and appears to have a normal urinary output, it is wise to document his status with a 24-hour urine collection and residual urine volume determination. If the patient demonstrates a residual of 100 ml or more, an indwelling catheter should be reinserted and urologic consultation sought. At this point in time, the patient may need a transurethral resection or therapy for neurogenic bladder.

If the diagnosis of prostatism can be made before abdominoperineal resection, preliminary transurethral resection can alleviate the problem of urinary retention.[8]

Simultaneous prostatectomy has been suggested during abdominoperineal resection, since a preliminary transurethral resection will delay subsequent rectal excision by 3 to 4 weeks.[9] Goligher[8] contends that the 3 to 4 week postponement will not greatly alter the final outcome, and retropubic prostatectomy and rectal excision done simultaneously greatly extend the period of recuperation.

SUMMARY

Abdominoperineal resection has long been recognized as a procedure with formidable morbidity. Through meticulous dissection, use of proper tissue planes, attention to location of urologic and neurologic structure, careful hemostasis, and appropriate postoperative measures, most of the complications outlined in this article can be avoided.

Should a complication develop, close surveillance and early recognition coupled with the appropriate management can still offer the patient a satisfactory recovery.

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Acute and Chronic Renal Failure

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Recent advances in medical technology in the past 15 years have provided the medical profession with a spectrum of patients with new and complex pathophysiology. One such outstanding category of patients is those with renal failure. Two basic mechanisms are responsible for the increasing frequency with which surgeons must deal with patients with renal failure. First, increasing numbers of patients are experiencing acute renal failure following major surgical intervention. A number of factors are responsible for this occurrence: patients who are at high risk for acute renal failure are being recommended for major surgery; certain complex operations are often associated with a high incidence of postoperative acute renal failure; and preoperative testing procedures frequently increase the risk of postoperative renal failure.

Prior to 1960, most patients with long-standing chronic renal disease died of chronic renal failure. The introduction of dialysis and renal transplantation into clinical medicine has, however, sustained life for a large population of such patients having otherwise fatal disease. There are presently greater than 8 million Americans with known renal disease, 35,000 of whom are receiving frequent dialysis to sustain life.[3] Many of these patients with organ failure require surgical intervention. Further, permanent vascular access and/or peritoneal cavity access is necessary for maintenance dialysis. Since alterations in metabolism occur in these patients,[27] a host of indications for repeated surgery and complications thereof occur specific to this population. Accordingly, the ensuing discussion will deal with two issues surrounding these patients. The first will discuss prophylaxis and management of postoperative acute renal failure and the second, surgical complications occurring in patients with chronic renal failure.

ACUTE RENAL FAILURE (ARF)

Postoperative ARF continues to result in a mortality of 50 to 70 per cent in spite of availability of interim dialysis for these patients.[6, 24, 60]

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The excessive mortality rate is not due to renal insufficiency itself but rather to the complications of ARF: gastrointestinal bleeding, systemic sepsis, pulmonary insufficiency, and wound dehiscence.[11, 20, 42] Irrespective of the causative mechanism, ARF is a self-limited disease which resolves approximately 7 to 28 days following the initial insult. For the most part, patients who survive the phase of ARF return to normal life style with quantitatively normal kidney function.[12] In contrast, the high patient mortality occurs during the interval of acute renal insufficiency and is due to the combined catabolic effects of surgery and the abrupt onset of uremia resulting in: a generalized accelerated rate of catabolism; suppression of nutrition; depressed resistance to infection; and impaired wound healing.[57] For these reasons, prophylactic considerations surrounding ARF should be directed toward: avoidance of risk factors; preoperative recognition of the patient who is high-risk; and maintenance of circulatory volume, state of hydration, and diuresis. Early recognition is the hallmark for effective treatment of impending postoperative renal failure. Once renal failure is detected, the therapeutic goals are to minimize fluid and electrolyte derangements, control hypertension, maintain optimal nutrition, anticipate complications, and treat uremia with aggressive dialysis.

The criteria for recognition and modalities of early treatment have recently been emphasized by Turcotte,[63] Danielson,[20] and Lucas[41] and will not be repeated herein. Instead, our emphasis is directed toward control of risk factors and maintenance of optimal nutrition during ARF.

Risk Factors

Irrespective of etiology, the common pathophysiologic mechanism which causes postoperative ARF is vasoconstriction of afferent arterioles in the kidney often referred to as vasomotor nephropathy.[48] The etiologic agents may be divided into four groups: (1) preoperative insult to the kidney, (2) hypovolemia, (3) hypotension, and (4) the presence of circulating endogenous or exogenous nephrotoxic agents (Tables 1 and 2).

Preoperative Status

History of Chronic Renal Disease. Certainly, patients with chronic renal disease are subject to ARF after less of an insult than is necessary to cause renal failure in patients with normal kidneys. Patients with a history of hypertension, diabetes mellitus, previous history of impaired kidney function or hematuria, and a familial history of kidney disease should be evaluated with detailed creatinine clearance prior to operative intervention. The presence of proteinuria, urinary casts, urinary tract infection, or pyuria should likewise be investigated. Note that renal function can be diminished to 25 to 40 per cent of normal in the absence of elevation of blood urea nitrogen and serum creatinine.

Table 1. *Preoperative Risk Factors*

CHRONIC DEHYDRATION
External fistulae
Diuretic therapy, especially
Hypertension
Congestive heart failure
Hepatic ascites
Pyloric stenosis
Esophageal stenosis
ACUTE DEHYDRATION
Burns
Trauma
Gastrointestinal bleeding
Pancreatitis
Peritonitis
Bowel obstruction
Sepsis
Acute intoxication
Toxic megacolon
HOSPITAL-INDUCED DEHYDRATION
Fasting
Osmotic diuresis (IVP, IVC, arteriography)
Arteriography (renal vasoconstriction)
Barium
Enemas
Cathartics
Nasogastric suction
HYPERTENSION
Signals renal disease
Abrupt control of systemic pressure
Long-acting antihypertensive agents (cardiovascular paralysis)

Table 2. *Nephrotoxic Agents*

ENDOGENOUS	EXOGENOUS
Fragmented formed elements in blood	General anesthesia
Atheromatous emboli	Renografin
Myoglobin	Aminoglycosides
Endotoxin	Cephaloridine, cephalothin sodium
Intravascular coagulation	Pentamidine
Fat emboli	Amphotericin B
Free hemoglobin	Rifampin
Renin-angiotensin-aldosterone	

Consequently, the presence of normal BUN and serum creatinine determinations does not mitigate against the presence of chronic renal insufficiency.

Hydration. Three categories of dehydration and consequent hypovolemia occur prior to surgery: chronic dehydration, acute dehydration—usually due to a recent insult (trauma, etc.), and hospital-induced dehydration. Chronic dehydration may be caused by longstanding colitis, external fistulae from the intestinal tract, or esophageal or pyloric stenosis. A particularly occult form of dehydration occurs in the form of diuretic therapy. Patients who are receiving diuretics for a long period of time for either hypertension, compensated congestive heart failure, or hepatic ascites exhibit a high incidence of postoperative ARF.[58] Not only is the chronic dehydration from diuretics a predisposing factor but patients with liver disease, hypertension, and heart failure all have a propensity for decreased renal perfusion even in the absence of a major stress such as surgery.

Acute hypovolemia prior to urgent or emergency surgery is generally due to an abrupt systemic insult. Third space losses due to peritonitis, pancreatitis, burns, and bowel obstruction are the most common causes of depletion of extracellular space fluids and of contracted intravascular volume. Major trauma results in third space losses as well as hypovolemia due to blood loss. A particularly important cause of relative hypovolemia is systemic sepsis. Sepsis cannot only cause hypovolemia but is also associated with decreased renal perfusion independent of hypovolemia.[44] The association of disseminated intravascular coagulation disorders in sepsis may independently cause renal failure due to the microangiopathic process within the kidney.

The alcoholic who experiences major blunt or penetrating trauma represents an intriguing pathophysiologic circumstance. Cirrhotic patients—particularly those with ascites—have a decreased renal blood flow.[5] Further, the presence of acute intoxication prior to trauma results in ethanol-induced dehydration. Thus, abrupt hypovolemia due to trauma in these patients represents the third additional insult contributing to dehydration, hypovolemia, and decreased renal blood flow.

Hospital-induced dehydration occurs in two types of circumstances. The patient who experiences an acute insult requiring emergency surgery may, in preparation for surgery, undergo a number of studies such as intravenous pyelography (IVP), arteriography, and intravenous cholangiography (IVC). Each of these tests is performed with hyperosmotic, radiographic solutions which effectively produce an osmotic diuresis and dehydration preoperatively.[23, 47] Moreover, direct introduction of hyperosmolar solutions into the renal arteries causes renal arterial constriction and a decrease in renal blood flow.[50] These patients often reach the operating room with the additive insult of these diagnostic procedures in addition to the initial acute event.

A second and often occult form of dehydration occurs in the patient who is being evaluated for 5 to 7 days prior to major surgery. These patients often receive a sequence of studies to include IVP, barium enema, upper gastrointestinal radiographic studies, proctoscopic examination,

and arteriography. In addition to the effects of IVP and arteriographic studies, a series of dynamic events occurs surrounding these procedures which results in dehydration. The patient is usually fasting and receives enemas before and after these procedures which effectively results in fluid losses; barium introduced into the lower gastrointestinal tract is hydrophilic and causes an efflux of fluid; cathartics are often given after barium is introduced into the upper gastrointestinal tract; and just as importantly, the patient is anorectic due to diet limitations and the stress of the sequential procedures. These patients are then brought to the operating room with a major fluid deficit. In our own series, we have found that a weight loss of 4 to 12 kilograms during 5 days of testing is not an uncommon event. Figure 1 delineates a 10 kilogram weight loss in one such patient being evaluated prior to major surgery. A less occult factor contributing to dehydration is the use of nasogastric suction in the preoperative period.

HYPERTENSION. Three distinct factors relating to hypertension increase the risk of postoperative renal failure. The presence of hypertension may be the result of chronic renal disease. Alternately, the hydrodynamic effects of hypertension on the kidneys may cause arteriolosclerosis and secondary deterioration in kidney function. A second circumstance is the patient who has uncontrolled hypertension and is in need of urgent or emergency surgery. Very often the blood pressure is brought under control preoperatively with quick-acting antihypertensive agents. This abrupt decrease in blood pressure to normal levels within 24 hours prior to surgery causes an abrupt decrease in creatinine clearance to as low as 50 per cent of previous values. This procedure represents serious renal insult and should not be performed unless absolutely necessary. Lastly, many patients come to the operating room after

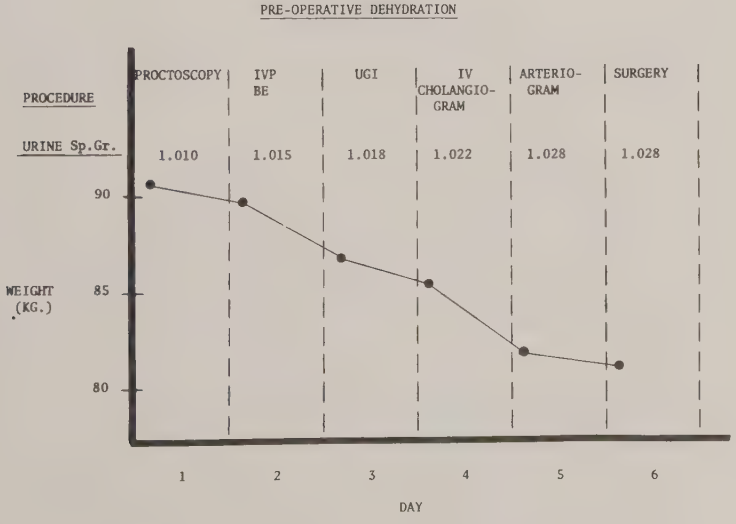

Figure 1. An example of 10 kg body weight loss after several days of diagnostic tests.

having been treated for long periods of time with antihypertensive medications. The effects of these agents often last for many days. Thus, a patient who experiences an abrupt blood loss intraoperatively may experience profound intraoperative or postoperative hypotension due to paralysis of the cardiovascular system induced by these long-acting agents.[37]

Preoperative Prophylactic Measures. All patients with a history or documentation of chronic renal impairment, patients with chronic dehydration, and patients who are dehydrated in the hospital due to extensive preoperative testing procedures should be well hydrated prior to operative procedures. Intravenous crystalloid replacement is recommended for 1 to 3 days preoperatively to expand both the vascular compartment as well as the extracellular space. Intravenous infusion of mannitol (12.5 gm) every 6 hours increases renal hydration. The acceptable end-point prior to surgery is an increase in the volume of urine to at least 60 ml per hour along with demonstration of dilute urine. In patients with acute insults such as trauma, gastrointestinal bleeding, etc., volume replacement with crystalloid, colloid, and blood components is indicated to maintain a large volume of urine output and a low specific gravity. Central venous pressure monitoring is necessary in these patients as an aid to adequate volume replacement prior to surgery. Emergency arteriography, IVP, etc., will artificially elevate urine volume and may alter specific gravity in these patients. To wit, the sine qua non of adequate volume replacement must not only be elevated urine output but an elevation in the central venous pressure.

Two factors must be kept in mind for the hypertensive patient who is being prepared for surgery. First, blood pressure should be brought under control during a period of 3 to 5 days prior to surgical intervention. The kidneys will therefore adapt to alterations in blood pressure and the patient will not enter the operating room with a seriously suppressed kidney function. Secondly, long-acting antihypertensive agents (aldomet, reserpine, etc.) should be discontinued and replaced with short-acting agents such as intramuscular hydralazine, intravenous diazoxide or nitroprusside.

Insults Surrounding Surgery

General anesthesia almost universally results in insult to the kidney. These agents cause a decrease in renal blood flow and an increase in vascular resistance within the kidney.[7,45] Abrupt volume losses intraoperatively such as occur in major vascular surgery, trauma with hemoperitoneum, and cardiac surgery must be kept to a minimum. Specific types of operations increase the risk of renal failure. Cardiopulmonary bypass,[25] abdominal aortic surgery,[43] and operations for long-standing obstructive jaundice[21] result in a high incidence of ARF. Patients with systemic sepsis, abdominal abscesses, hepatic sepsis, and septic abortions all are at risk for renal failure due to decreased renal blood flow caused by shock, endotoxic vasoconstriction,[44] and occasionally intravascular coagulopathy.[18]

Trauma patients with extensive crushing injuries, rhabdomyolysis,

and myoglobinemia or those with long bone fractures and fat emboli are at risk for ARF.[9, 16, 61] Rapid hemolysis of blood due to blood transfusion reactions, septic hemolysis, or transfusion of debilitated red blood cells results in hemoglobinemia providing an additional insult to the kidneys.[54]

Patients with sepsis very often come to operation while receiving nephrotoxic antibiotics. Most commonly used are the aminoglycosides including gentamycin and kanamycin. Rarely, renal failure may be caused by cephaloridine or cephalothin sodium.[14] Less commonly used agents which contribute to acute renal failure include colistin, neomycin, pentamidine, and amphotericin B. When nephrotoxic antibiotics must be used, one should maintain volume replacement and liberal use of mannitol to enhance excretion. Further, renal function should be evaluated on a daily basis in order to appropriately alter the daily dose of the antibiotic to avoid toxic levels in the serum. Table 2 provides a list of nephrotoxic agents both of endogenous and exogenous origin which contribute to renal insult surrounding major surgical interventions.

Management of Acute Renal Failure

Therapeutic goals in the management of ARF are not only directed to minimize uremia, hyperkalemia, and fluid derangements but also to avoid the complications which effect high mortality. Since impaired wound healing, resistance to infection, and stress gastrointestinal bleeding are all a result of impaired nutrition, we feel strongly that the hallmark for treatment surrounds maintenance of optimum nutrition.

Nutritional Therapy

Protein restriction is necessary in ARF to avoid rapid rise in BUN.[62] Until recently, patients have experienced a major caloric deficit on this regimen. In the last few years we and other investigators have used a combination of amino acids and hypertonic glucose intravenously to provide improved nutrition. A combination of 250 ml of a solution of eight L-essential amino acids (FreAmine-E, McGaw) plus 750 ml of 50 or 70 per cent dextrose monohydrate solution provides nearly 1.5 cal per ml in the resulting solution. This parenteral therapy is instituted through a closed system and infused through a central vein (usually subclavian catheterization). The amino acids are utilized to synthesize protein and recycle the available urea into protein synthesis.[20] The rate of rise of urea within the blood is suppressed when compared to patients without this therapy.[1, 2] Serum albumin is maintained at higher levels than in the absence of treatment. In order to supply 3000 to 4000 calories per day, approximately 2000 to 3000 ml of fluid must be administered. One of the major functions of dialysis is, therefore, to minimize volume overload during nutritional therapy. When the patient is able to tolerate oral alimentation, the amino acid regimen may be given orally or by a constant infusion through a nasogastric feeding tube in the form of Aminaid.

Dialysis, Fluid and Electrolyte Balance

Classically, a strict regimen of fluid and salt restriction was the primary treatment of ARF. However, in the presence of zealous, nutritional replacement, volume overloading will be a major consideration in the absence of aggressive early dialysis. As a result, dialysis should be instituted concomitantly with alimentation in the early phases of ARF. Hemodialysis is preferable under most circumstances since it is more effective in removing excess fluid than peritoneal dialysis. Frequently, dialysis is employed either every day or every other day. Patients who have septicemia or an unstable cardiovascular status, those in danger of postoperative bleeding when heparinized, who have a bleeding tendency, or who have head injuries should have peritoneal dialysis. Peritoneal dialysis will effectively remove excess salt and water when a hypertonic solution is infused peritoneally (4.5 per cent dextrose solution, Dianeal, Travenol). Aggressive dialysis will prophylactically treat insidious onset of hyperkalemia as well as metabolic acidosis, each of which accompanies ARF. It should be noted that judicious use of nasogastric suction will also provide an adjunct in the treatment of metabolic acidosis and in removing excess fluid volume and potassium. Daily weight of the patient should be recorded and monitored carefully. Allowing for an initial weight gain due to third space accumulation of salt and water postoperatively, management with intense nutritional support will result in a slight weight loss (less than 0.3 kg per day). This therapeutic approach in our own series has brought the incidence of stress gastrointestinal bleeding and sepsis to a minimum. It has decreased patient mortality to approximately 25 per cent. We have experienced no episodes of wound dehiscence.

Complications

Sepsis

Investigation for sepsis is a necessity in patients with ARF. Frequent blood cultures and urine cultures will improve early diagnosis. Prophylactic treatment with antibiotics is not advised since overwhelming infections with opportunistic bacteria and fungi will often result. Once ARF is established, the central venous pressure and Foley catheter should be removed to avoid these sources of contamination. Abdominal drains must be treated with strict aseptic technique and be removed as soon as feasible. Poorly drained areas and developing abscesses must be diagnosed and treated with adequate drainage as early as possible. Devitalized tissue must be removed. In cases of gangrene of the lower extremities, early amputation is advised. When sepsis is documented, specific antibiotics should be used. Therapeutic dosage must be altered to avoid toxic levels in renal failure. The reader is referred to Antibiotic Therapy charts for patients in renal failure.[6] Nephrotoxic antibiotics should be meticulously avoided unless absolutely necessary.

Gastrointestinal Bleeding

The occurrence of stress ulcer bleeding in ARF carries an 80 per cent mortality.[20] It probably results from the effects of poor nutritional support and ischemic insults to the gastrointestinal mucosa. Nonoperative measures such as instillation of iced saline with or without norepinepherine and intense antacid therapy through a nasogastric tube represent initial management. Infusion of pitressin either through a peripheral vein or directly to an appropriate visceral artery has proved to be a major factor in halting bleeding.[13] Operative intervention is usually avoided unless persistent bleeding occurs after transfusion of 5 units of blood. Truncal vagotomy, transgastric ligation of bleeding points, and pyloroplasty are preferred therapy in most patients.

COMPLICATIONS OF SURGERY IN CHRONIC RENAL FAILURE

Surgical procedures and their complications in chronic renal insufficiency may be divided into two groups. The first are common operative procedures performed in patients with chronic renal failure. The second are complications inherent in chronic renal failure which require surgical intervention.

Common Surgical Procedures in Renal Failure Patients

Invariably, the most commonly performed operations for patients with chronic renal insufficiency are *access* procedures for dialysis. Temporary or permanent peritoneal catheters are placed for peritoneal dialysis. External arteriovenous cannulae or, alternately, internal arteriovenous fistulae are constructed to provide angioaccess for hemodialysis. The next most common procedure performed for chronic renal failure is nephrectomy—usually for hypertension or in preparation for renal transplantation.

Temporary Peritoneal Catheters

Temporary peritoneal catheters are inserted into the peritoneal cavity for intermittent dialysis either because dialysis is urgently required or when it is estimated that dialysis needs will be infrequent. Temporary catheters are placed percutaneously in the midline immediately below the umbilicus. The catheter is inserted blindly with a metal trocar which penetrates the peritoneal cavity and is withdrawn after insertion of the catheter. The most common complications are related to insertion. Trocar catheterization may cause bleeding, cannulation of abdominal viscera, or inadvertent introduction of the catheter into the preperitoneal space. The next most common complication is bacterial contamination of the peritoneal cavity during catheter usage. In our series of 520 temporary catheterizations over a 2-year period, 9 per cent

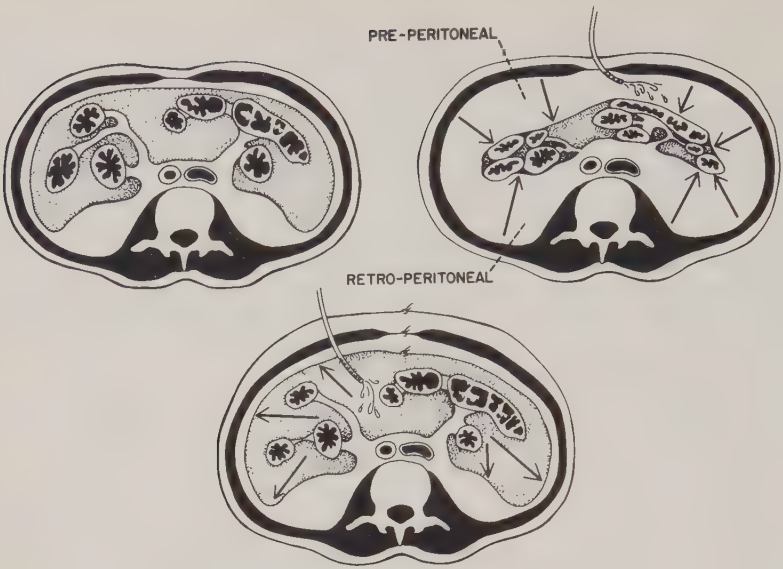

Figure 2. Inadvertent extraperitoneal cannulation.

experienced complications. Approximately one half of these required surgical intervention while the remaining 50 per cent were due to peritoneal bacterial contamination. Indications for emergency surgery were return of gross blood or succus entericus from the peritoneal catheter, abrupt onset of watery diarrhea, return of turbid fluid with particulate matter, or abrupt onset of peritonitis within hours of insertion of the catheter.

Bleeding. Hemorrhage following peritoneal catheter insertion usually arises from the abdominal wall and the base of the mesentery. However, we have observed inadvertent cannulation of iliac artery or vein, and impalement of a pelvic kidney as the source of bleeding.

In contrast, it is not uncommon to find blood-stained fluid return immediately after insertion of the peritoneal catheter. Most physicians will begin peritoneal dialysis and frequently examine the fluid for presence of blood. Each dialysis cycle should subsequently have less and less blood-tinge to the fluid. If blood persists in the fluid for greater than 12 hours, or if it increases in magnitude, exploratory laparotomy is recommended.

A particularly occult form of bleeding occurs when the catheter is inadvertently placed in the preperitoneal space. In this circumstance, the initial dialysate return will have little or no blood present. Subsequent dialysis cycles will have an increasing amount of blood efflux. The mechanism of bleeding is progressive dissection of the peritoneal lining away from the preperitoneal and retroperitoneal soft tissue, resulting in injury to multiple small venous channels especially in the pelvic area. In the presence of constant irrigation with a nonviscous, nonclotting fluid (dialysate), the bleeding persists without clotting. Figure 2 delineates the anatomy during dissection of the preperitoneal

space in the upper right drawing. We have experienced two patients with this complication. Upon entering the abdominal cavity, the peritoneum is found to be dissected far away from the preperitoneal and retroperitoneal soft tissues. Large amounts of blood and dialysate fluid are present. Effective treatment consists of removal of the malplaced catheter and replacement of a permanent peritoneal catheter into the true peritoneal cavity. Reinstitution of peritoneal dialysis is an essential part of effective treatment. The infusion of dialysate solution into the true peritoneal cavity will compress the peritoneal lining against the soft tissues of the preperitoneal and retroperitoneal space, thus effectively producing tamponade of the multiple bleeding sites. The mechanism is illustrated in the lower drawing in Figure 2. We consider it important to continue peritoneal dialysis for 7 days to produce effective hemostasis. In one of our patients, the dialysis was discontinued 24 hours after surgery. The patient experienced recurrent bleeding into the retroperitoneal space and died of blood loss.

Visceral Cannulation. Return of succus entericus in the catheter indicates cannulation of the bowel. Some investigators have treated this complication by removal of the peritoneal catheter and institution of systemic antibiotics.[34] Our experience has indicated that this form of therapy is not adequate. Very often these patients have many adhesions which cause compartmentalization of the peritoneal cavity from previous peritoneal catheters. As a result, leakage from the small bowel does not invariably result in overt peritonitis. However, loculated abscesses occur within pockets in the peritoneal cavity. This event results in chronic debilitation of the patient and search for the cause of fever at a later date. In our opinion, return of small bowel contents following catheter insertion is an indication for immediate surgery and repair of the damaged bowel.

Abrupt onset of peritonitis within hours after cannulation indicates soilage of the peritoneal cavity. In contrast, bacterial contamination of the peritoneal cavity from dialysis catheter usage will not occur for 48 hours after contamination. Immediate laparotomy is indicated in the former circumstance and invariably partial cannulation of small or large bowel is present. Abrupt onset of diarrhea or return of particulate matter in the dialysate fluid indicates cannulation of the bowel (usually colon) and requires immediate surgical intervention. Early diagnosis and operative intervention for cannulation of the small or large bowel is adequately managed by simply oversewing perforations and debriding devitalized tissue. We recommend insertion of a permanent peritoneal catheter at the time of abdominal exploration. Reinstitution of a peritoneal dialysis with antibiotics in the perfusate is an effective aid in sterilization of the peritoneal cavity.

Abrupt onset of "polyuria" after peritoneal catheterization indicates cannulation of the urinary bladder. This complication does not require operative intervention. Foley catheter decompression of the urinary bladder combined with removal of the peritoneal catheter provides effective treatment. Foley decompression should be maintained for 3 to 5 days. Prior to insertion of temporary catheters, the physician should insure that the urinary bladder is empty. Table 3 lists the findings, the in-

Table 3. *Complications from Insertion of Temporary Peritoneal Catheters*

FINDINGS	PATHOLOGY	TREATMENT
Bleeding	Vascular injury Preperitoneal catheterization	Surgery
Succus entericus	Small bowel cannulation	Surgery
Early peritonitis	Bowel cannulation	Surgery
Diarrhea; particulate matter (fecal material)	Large bowel cannulation	Surgery
Polyuria	Urinary bladder cannulation	Foley catheter

juries noted, and the treatment indicated for injuries due to insertion of temporary peritoneal catheters.

Contraindications. Complications secondary to insertion of temporary peritoneal catheters can be avoided by considering certain contraindications. Patients who have had previous abdominal surgery, prior peritoneal catheterization (either temporary or permanent), demonstrable organomegaly, profound obesity, or abdominal distention will experience a higher incidence of traumatic complications due to insertion of the catheter. If peritoneal dialysis is indicated in these patients, insertion of a permanent peritoneal catheter under direct vision is recommended.

Permanent Peritoneal Catheters

Permanent peritoneal catheters are inserted in patients in whom repeated peritoneal dialysis will be required. There are two Dacron felt cuffs along the length of the catheter to stimulate tissue ingrowth from the catheter tract. Proper placement of the permanent catheter is indicated in the left drawing in Figure 3. Notice that the deep Dacron cuff

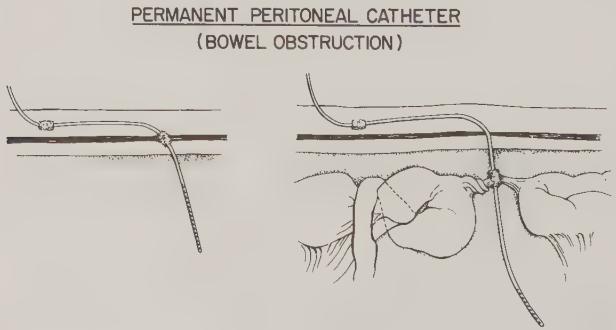

Figure 3. Bowel obstruction caused by malplaced internal Dacron felt cuff.

is at the fascia of the abdominal wall and the superficial Dacron cuff is in the subcutaneous layer along the subcutaneous tunnel. Permanent peritoneal catheters are placed under direct vision using local anesthesia. Complications arising from permanent peritoneal catheters are, for the most part, delayed in onset.

Infection. The most common complication arising from chronic dialysis is the gradual onset of peritoneal irritation and a cloudy return of dialysate. If fluid contains numerous white blood cells, treatment should consist of antibiotic therapy systemically as well as in the dialysate. Infections are usually due to gram negative rods. Most antibiotics cross the peritoneal barrier and will contribute to extracellular blood levels. The dosage of antibiotic must be calculated for the dialysate fluid. Therapy should continue for at least 3 to 5 days following absence of peritoneal findings and in the absence of white blood cells in the dialysate efflux.

Bowel Obstruction. Bowel obstruction due to peritoneal catheter usage is an uncommon complication. We have experienced this complication under two circumstances. First, debilitated patients may have low grade inflammation and infection within the peritoneal cavity in the absence of physical findings. Chronic inflammation results in multiple adhesions which may contribute to bowel obstruction. The second reason for bowel obstruction is represented in the right diagram of Figure 3. Improper technique may result in placement of the deep Dacron cuff within the peritoneal cavity. Dacron stimulates fibrous adhesions which on three occasions in our center have caused a closed loop bowel obstruction as shown in Figure 3. It is important to transfix the deep Dacron cuff to the fascia at the time of catheter placement to avoid this complication. It should be noted that bowel obstruction may mimic bacterial contamination of the peritoneal cavity from catheter usage. Gradual onset of abdominal distention with or without signs of peritonitis may indicate bowel obstruction with or without strangulation. Therefore, the presence of these findings during peritoneal dialysis should be accompanied by an intense physical examination along with radiography of the abdomen. Early operative intervention for bowel obstruction provides effective treatment.

Retained Catheter. Occasionally, a peritoneal catheter will be lost within the peritoneal cavity. This is usually the result of faulty removal of the catheter. Since the catheter is transfixed at the fascia, permanent peritoneal catheters should be removed under direct vision in the operating room. Either a patient or a physician may pull out the permanent peritoneal catheter intentionally or inadvertently. Invariably, the catheter is transected at the level of the fascial transfixion suture. Operative removal of the remaining catheter should be performed when this circumstance occurs. Retained catheters result in chronic peritoneal irritation, abscess formation, and debilitation of the patient. This complication will be discussed in a later section.

External Arteriovenous Cannulae

The external arteriovenous cannula described by Quinton et al.[51] provides an access for acute hemodialysis by placement of Teflon can-

nulae and Silastic extension tubing into a peripheral artery and vein. External catheters are used less commonly today due to their high incidence of thrombosis,[39] infection,[52] and catheter dislodgement with subsequent hemorrhage. Each of these complications may occur in the immediate postoperative period or after the catheter has been in use for some time. The average longevity of these catheters ranges from less than 1 month to approximately 12 months.[66] A few investigators have reported longer life in these catheters.[38]

Thrombosis. Thrombosis is the result of technical error, low cardiac output, or high-grade arterial or venous obstruction unnoticed at the time of operation. If the patient is experiencing a low cardiac output syndrome, it is futile to thrombectomize or replace the catheter. In the absence of low cardiac output, the catheter should be replaced in the operating room. Malalignment of the catheter tip within the vessels and damage to vascular intima are the most common causes of thrombosis. During reoperation, attention to straight alignment of catheter tips within the vessels is essential. Excellent pulsatile flow must be achieved from the arterial side and lack of venous resistance must be documented through the venous catheter. Should either of these events be questionable, intraoperative arteriogram and/or venogram should be obtained in search of proximal vascular stenosis.

Thrombosis during usage may be either due to an obstruction on the arterial side or an increment in the venous outflow resistance. Initial management is Fogarty thrombectomy. In the event of repeated thromboses, arteriography and venography should be performed. The occurrence of atheromatous plaque or intimal fibrosis on the arterial side will necessitate replacement of the arterial cannula proximal to the obstruction. The occurrence of segmental narrowing of the vein can be treated with replacement of the venous catheter proximally. Long segment stenosis of the vein implies failure of the catheter on the venous side. Repeated clotting of external cannulae in the absence of the demonstrable hemodynamic obstructions may be treated with long-term anticoagulation therapy.[66]

Infection. Onset of infection in the immediate postoperative period is signaled by swelling and tenderness of the operative incision, and purulent discharge surrounding the catheter exit sites. The catheter should be clamped to reduce spread of infection through the systemic vascular circuit. Systemic antibiotics should be used. Two to 3 days following institution of therapy the catheter should be removed. Latent infection in external cannulae may present with a purulent drainage, but serous or bloody discharge from the catheter site may also represent infection. Since most infections are due to staphylococci, systemic vancomycin has been recommended as the agent of choice.[26] If the infection does not resolve rapidly, the cannula should be clamped and removed 3 days later. Once bleeding occurs from the cannula, we prefer to remove the cannula since the infection has more than likely involved the wall of the blood vessel. If the patient requires dialysis, a peritoneal catheter should be inserted and used until infection is eradicated.

Catheter Dislodgement. Postoperative dislodgement of external catheters with resulting hemorrhage should be treated with pressure

dressing, blood replacement, and operative ligation of the open vessel. At reoperation, replacement of another catheter may be performed if sterility has been insured. Latent dislodgement of catheters may be due to the trauma from recurrent use, but may reflect infection at the catheter exit site. For this reason it is recommended that the dislodged catheter be abandoned and the vessels ligated.

Skin Erosion. An infrequent complication from external cannulae is erosion of the skin. The phenomenon is due to pressure on the skin superficial to the portion of cannula immediately subjacent to the skin. The presence of necrosis indicates potential infection. The catheter should be removed and the necrotic skin excised. Avoidance of this complication is accomplished by placement of the catheter tip well beneath the subcutaneous layer.

Internal Arteriovenous Fistula

Internal arteriovenous fistulae are most often performed by direct anastomosis of a peripheral artery to a peripheral vein. The most common site is the radial artery to cephalic vein in the forearm popularized by Brescia et al.[10] If proper operative technique is employed, complications are rare.[35] Thrombosis occurs in less than 5 per cent of patients.[15] If thrombosis does occur at the anastomotic site, the only recourse is reexploration and construction of an arteriovenous fistula immediately proximal to the initial fistula. Latent thrombosis of arteriovenous fistulae after frequent use may be due to fibrotic stenosis of the vein or acute thrombosis secondary to over-zealous pressure dressing after dialysis. Thrombectomy is indicated. It is useful to obtain an operative angiogram to insure that severe fibrotic stenosis has not occurred. A short segment of stenosis may be treated with angioplasty but a long segment stenosis is generally not salvageable.

Infection. Postoperative infection rarely occurs following construction of arteriovenous fistulae. Many investigators use prophylactic, single-dose vancomycin preoperatively.[46] Latent infection occurs at the site of venous puncture for hemodialysis. Antistaphylococcal antibiotics and immobilization of the forearm will eradicate most infections. If infection persists, perforation of the vessel and bleeding may occur. Direct pressure should be applied and the proximal and distal vessels must be ligated, sacrificing the fistula to avoid serious hemorrhage.

Venous Hypertension. When side-to-side anastomoses are performed, venous hypertension in the hand occasionally occurs. Venous hypertension most often presents with distended veins on the dorsum of the hand. Rarely, edema followed by changes comparable to venous stasis in the lower extremity occurs. When these changes are present, an angiogram of the distal venous segment should be obtained to localize the venous branches to the hand. These branches are easily ligated and effectively reverse the process.

Aneurysms. Aneurysms of the venous segment are uncommon. They are probably due to weakening of the venous wall at puncture sites. Morbidity from these aneurysms is minimal. Mural thrombi have only rarely been reported to produce pulmonary embolism. Enlargement of the aneurysm causing pressure necrosis on the overlying skin is dan-

gerous and may result in an explosive bleeding episode. Resection of the aneurysm along with the compromised skin must be performed under these circumstances.

Heart Failure. Very rarely, congestive heart failure can be attributed to the left to right flow in a peripheral arteriovenous fistula.[33] More commonly, large arteriovenous fistulae constructed with graft material (saphenous vein, bovine graft, etc.) may produce symptoms of congestive heart failure.[65] Under these rare circumstances, operative constriction of the graft to decrease the volume flow may be helpful. Intentional stenosis, however, increases risk of thrombosis.

Ischemia. Very rarely, arteriovenous fistula causes symptoms of ischemia in the distal extremity.[60] When these symptoms do occur, ablation of the fistula along with maintenance of arterial integrity reverses symptomatology.

Nephrectomy for Hypertension

Numerous patients with chronic renal failure experience hypertension. Blood pressure in the majority of these cases is volume-related. Astute reduction in extracellular volume during dialysis will control hypertension. Occasionally, hypertension is caused by the renin-angiotensin mechanism. When pressure is not controlled by volume depletion or antihypertensive agents, bilateral nephrectomy is often performed.[17, 40] A rather unique postoperative complication occurs in these patients. Postoperative profound hypotension has been reported and has caused mortality.[53] The mechanism appears to be an abrupt increase in the size of the vascular compartment due to acute renin withdrawal. In our own experience, patients have required as much as 10 liters of fluid intravenously in the first 24 hours after surgery to avoid hypotension. Hypotension from acute renin-withdrawal is avoided by hydration of the patient preoperatively followed by intense hydration of the patient while monitoring the central venous pressure postoperatively. The average postoperative fluid replacement in our group of patients has been 250 to 350 ml per hour for the first 24 hours.

COMPLICATIONS OF CHRONIC RENAL FAILURE REQUIRING SURGERY

Secondary Hyperparathyroidism

Chronic renal insufficiency invariably produces secondary hyperparathyroidism.[36] Moderate deterioration in kidney function is associated with elevated blood parathormone levels.[49] Further, malabsorption of vitamin D from the intestinal tract occurs as azotemia progresses.[22] When kidney function is reduced to 25 per cent of normal, phosphate retention ensues. A reciprocal suppression in serum calcium stimulates the parathyroid glands. Chronic chief-cell hyperplasia persists and becomes marked as chronic renal failure continues. Resorption of calcium from bone increases the calcium-phosphorus product in the extracellular space. When serum calcium times serum phosphorus reaches a value of

approximately 75, precipitation of calcium phosphate crystals occurs in soft tissues. Parathyroidectomy is indicated when patients experience bone pain, when demonstration of soft tissue calcification occurs, and when calcium-phosphorus product is maintained persistently at dangerously high levels.

Disagreement persists as to the optimum procedure during parathyroidectomy. Certain investigators perform total parathyroidectomy to avoid the continued stimulation of remaining glandular tissue which occurs in subtotal parathyroidectomy.[56] Other investigators perform a total parathyroidectomy with parathyroid autotransplant.[4] Autotransplantation of parathyroid tissue into muscle provides an easily accessible area for reoperation if removal of more parathyroid tissue is necessary at a later date. Further, the procedure provides retained parathyroid tissue for the patient in the event that he or she receives a kidney transplant at a later date. Total parathyroidectomy results in a difficult problem at the time of renal transplantation.[28] After renal transplantation, phosphorus is excreted. Reciprocal elevation in serum calcium does not occur in the absence of parathyroid tissue. Tetany will ensue if untreated. While subtotal parathyroidectomy during renal insufficiency appears to be the safest procedure, reoperation to remove additional parathyroid tissue has the inherent hazard of injury to the recurrent laryngeal nerve. Our present trend is to perform total parathyroidectomy with parathyroid autotransplant for patients in chronic renal failure.[29]

"Dialysis Ascites"

A number of patients with chronic renal insufficiency develop ascites in the absence of demonstrable hepatic disease.[19, 31, 55, 65] Many of these patients also experience weight loss, rapid debilitation, and occasionally intermittent fever. Until recently, the term "dialysis ascites" has been used to identify patients who have no other apparent cause for the peritoneal accumulation of fluid. Recently, investigators have postulated the presence of a renal factor responsible for the ascites.[55] Bilateral nephrectomy has been reported to reverse the muscle wasting and halt the reaccumulation of ascites. We have therefore performed bilateral nephrectomy under these circumstances for a number of our patients. Reversal of the process occurred with no apparent etiology in approximately 40 per cent of patients. In the remaining 60 per cent, exploratory laparotomy revealed a cause for ascites. The inflammatory focus within the abdominal cavity was either due to a previous percutaneous kidney biopsy or prior peritoneal catheterization for dialysis. Perinephric abscess was the result of a prior kidney biopsy. The remaining patients had occult abdominal abscess secondary to previous peritoneal catheterization, to a previous bowel perforation (presumably from temporary peritoneal catheter insertion), or a retained peritoneal catheter producing inflammation and infection. These data indicate that all patients with chronic renal insufficiency who have a triad of ascites, lethargy, and muscle wasting should undergo laparotomy. If cause is not found for the ascites and chronic debilitation, then bilateral nephrectomy should be performed.

Hypersplenism

Hypersplenism reportedly occurs in approximately 20 per cent of patients with chronic renal insufficiency.[8] It is well known that renal failure is associated with a depressed half-life of formed elements in the blood. Further, the bone marow is often suppressed to a greater or lesser degree. The spleen becomes hyperactive in removing deteriorated elements from the blood. Occasionally, extramedullary hematopoiesis occurs within the spleen. Resultant hypersplenism may occur producing pancytopenia and the need for frequent blood transfusions.[32] The presence of an hematocrit persistently less than 20 per cent associated with moderate leukopenia, thrombocytopenia, and the need for blood transfusions should raise the diagnostic possibility of hypersplenism. The absence of an enlarged spleen on sulfur colloid scan does not exclude the diagnosis. Exploratory laparotomy and splenectomy will often delineate an enlarged spleen with extramedullary hematopoiesis. Postoperatively, these patients will have an elevation in all blood formed elements and a decrease in the need for blood transfusions.[32]

Uremic Pericarditis

The presence of uremic pericarditis suggests less than optimal dialysis therapy. Patients experiencing chest pain are found to have pericardial friction rub along with changes on the electrocardiogram. Appropriate treatment is aggressive dialysis therapy. When pericarditis does not improve rapidly the patient must be observed for early signs of pericardial tamponade. If this disease progresses, urgent pericardiectomy through a left anterior thoracotomy should be performed before overt tamponade ensues. A wide pericardial window draining into the left chest cavity is sufficient management in the majority of cases.

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Total Parenteral Nutrition

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The technique of total parenteral nutrition has frequently been referred to as "hyperalimentation." For purposes of clarity, the use of the term "hyperalimentation" will be avoided in this article and the words "total parenteral nutrition" or "TPN" will be used to designate the technique whereby an attempt is made to provide for all nutritional needs for a prolonged period of time using a nonenteric route, generally intravenous. When the intravenous route is used as the primary source of calories, amino acids, electrolytes, and minerals, while partial intestinal function allows for supplemental enteric feedings, the intravenous component of the total nutritional program is referred to as "parenteral nutrition." The word "starvation" is commonly defined as "long-continued deprival of food."[17] In the present context, the word is subdefined to describe the state of the body which exists during a negative caloric or nitrogen balance for an interval in excess of 7 days.

A complete discussion of the metabolism of human starvation, the effects of the stress response, and the science of intravenous nutrition is well beyond the intended scope of this section. Many current books have been devoted to these topics, and the reader is referred to such texts for an amplification of both concepts and details in this rapidly expanding field of medical practice.[3, 23, 28, 36, 49, 52] The very unique needs of infants and children are not included in the subsequent discussion.

THE GENERAL RESPONSE TO FASTING AND STARVATION

Daily nutritional needs for healthy adults vary widely and generally reflect the individual's need for energy to maintain body temperature, carry on metabolic processes, and support physical activity. When daily

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Table 1. *Approximate Tissue Fuel Composition of Normal Man**

FUEL	Kg	CALORIES
Fat (adipose triglyceride)	15.0	141,000
Protein (mainly muscle)	6.0	24,000
Glycogen (muscle and liver)	0.225	900
		165,900

*As modified from Cahill.[13]

energy needs are not met because of either increased energy expenditure (fever, stress, increased physical activity) or decreased energy intake (fasting, starvation), available body substrates are used as energy sources to meet the daily requirements. The total available tissue substrates (protein, carbohydrates, fat) of an average 70 kg adult male are shown in Table 1.[13]

It is clear from a consideration of Table 1 that man has only two major fuel depots which enable him to survive periods of fasting beyond 24 hours. These fuel depots are fat and protein. The proteins, being essential to all vital functions of the body, are used as an energy source only at major expense to the body, as all protein consumed as energy is function lost to the body. Thus the final goal of the many metabolic adaptations the body makes during starvation is the conservation of body protein at the expense of body fat.[13, 46]

As starvation becomes prolonged, negative caloric and nitrogen balances persist until either sufficient nutriment is provided to reverse the balances or major proportions of body proteins are lost. Should the progressively starved individual survive the ravages of unhealed wounds and loss of immunocompetence, a final endpoint is reached when approximately one-third of body protein is lost. At such a major level of protein loss respiratory musculature commonly fails, and a pneumonic death becomes imminent. Under conditions of stress (sepsis, trauma, burns, surgery), daily caloric and protein expenditures may be several times the usual basal requirements. This effectively shortens the interval of time during which life-threatening catabolic events can occur.[13, 46]

PATIENT SELECTION

All ill patients have definite daily energy and nutritional needs, and with few exceptions these needs exceed those of the same individual when in good health. While body reserves may temporarily meet these needs in the acute situation, these reserves can become rapidly depleted, leaving the patient both ill and starved. The weight of accumulated evidence must lead us to the conclusion that the ill and starving patient will have a much improved chance of recovery if the starvation component is eliminated.[5, 7, 15, 35, 37]

Bedside recognition of the early stages of starvation is difficult

because of our inability to measure body protein losses and remaining protein reserves accurately. Variables in therapy such as the degree of blood volume replacement and the intermittent administration of protein-containing fluids such as albumin only compound the problem of nutritional assessment. Recent work by Blackburn, Bistrian, and others has led to a method of profiling the nutritional and metabolic status of hospitalized patients using bedside and laboratory measurements.[4, 6, 8] In the absence of such a profile, our recent baseline criterion for the initiation of aggressive nutritional support has been a 7-day (or longer) interval of negative caloric and nitrogen balance with no imminent return of normal gastrointestinal function. This baseline time interval has been shortened only in the obviously hypermetabolic patient such as the patient suffering from major burns or multiple injuries.

ROUTES OF THERAPY

Once the need for special nutritional support has been established, the physician must weigh the risks, benefits, and efficiency of three possible routes of nourishment—oral (or oral-enteric), enteric, and parenteral (Fig. 1). For those patients with a functional gastrointestinal tract, supplemental feedings with or without the addition of elemental diets may be sufficient. If an oral, esophageal, or gastric impediment to feeding exists, a nasogastric, gastric, or jejunal feeding tube may be indicated. When tube feedings are used, the risks of aspiration, metabolic abnormalities, and nonabsorption of nutrients must always be anticipated. Most of these risks can be minimized by the gradual introduction

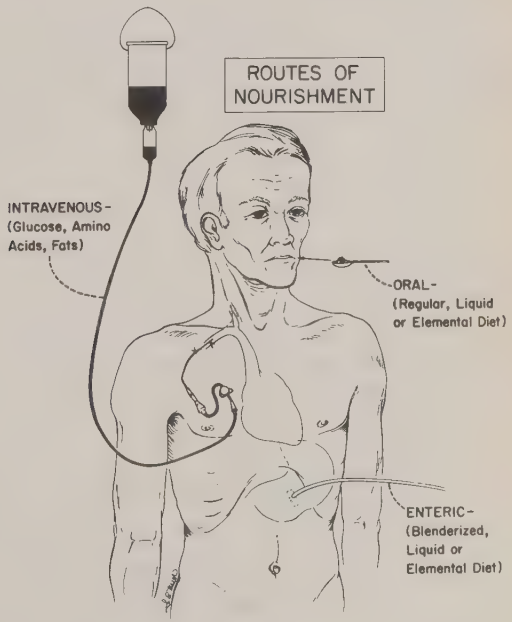

Figure 1. Three possible routes of nourishment in man.

of feedings with advancement after patient tolerance is demonstrated. The use of elemental diets can be a major advantage when digestive capacity is uncertain.[11, 12, 32]

Total parenteral nutrition is indicated in those patients who are in a negative caloric and nitrogen balance and whose gastrointestinal function is insufficient to reverse these balances over a reasonable interval of time. For the previously well nourished adult, a reasonable interval can be 7 to 11 days. For a prestarved adult, or an adult with increased energy and protein needs, the 7-to-11 day interval may be much too long, and aggressive nutritional therapy must begin before the 7th day.

In some patients the adequacy of tube feedings remains doubtful after a 7 day challenge using the enteric route. Nitrogen balance studies, while helpful in this situation, are not always available. When an experienced TPN team is available the intravenous route can be safely and efficiently used as the primary route for the administration of calories, nitrogen, and electrolytes, and the oral or enteric route used to supplement the patient's water, vitamin, fat, mineral, and trace element needs. The advantages of this approach are threefold: (1) the intravenous route gives the physician more precision in determining and controlling actual caloric and nitrogen intake; (2) the risks of intravenous nutrition in the hands of an experienced team can be so low as to be less than that of oral or tube feedings of borderline adequacy; and (3) patient nutritional and metabolic reserves can be more rapidly repleted, providing a more adequate cushion of reserve should unforeseen challenges (such as surgery or infection) occur during hospitalization.

The importance of an experienced TPN team cannot be overemphasized. Several studies have demonstrated catheter infection rates in excess of 15 per cent when catheter care is not provided by trained personnel.[16, 29, 47] Metabolic problems can be both numerous and serious when therapy is undertaken by individuals unfamiliar with the many potential hazards of the TPN system.[19, 26, 52] The TPN team has as major responsibilities the care of intravenous access routes, the maintenance of metabolic flow sheets, and the training of all personnel in contact with the patient of the special needs of the patient receiving TPN. The size of a TPN team will vary with the needs of each institution, but at minimum should consist of physician, nurse, and pharmacist.

All intravenous nutritional solutions are prepared by the pharmacy using a sterile, closed technique within a laminar air-flow hood. Additives to the basic solutions other than electrolytes and vitamins are avoided, thereby eliminating the problems of inactivation of both additive and solution.

ESTABLISHING THE INTRAVENOUS ROUTE

Successful TPN requires that sufficient nonprotein calories be given to exceed the patient's daily caloric expenditure. Achieving such high caloric intakes in critically-ill adults for prolonged intervals requires the use of glucose-amino acid solutions which are hyperosmo-

lar in relationship to blood plasma. These solutions, when administered intravenously, must be rapidly diluted to avoid the complications of phlebitis and hemolysis. Adequate dilution has been demonstrated in both the superior and inferior vena cava; using the superior vena cava reduces the risk of catheter dislodgment or infection. Access to the superior vena cava is usually obtained by subclavian venipuncture, though an upper arm cephalic vein cutdown can provide an alternate route in the patient with a tracheostomy or other potentially infected neck wound.

The placement of central venous catheters using the subclavian approach is basic to the technique of TPN and has been described by many authors.[18, 22, 50] Safe subclavian catheter placement requires meticulous attention to details of patient preparation (including blood volume replacement and bedside positioning), use of anatomic landmarks and sterile technique, and care in the handling of needles and catheter if the numerous potential complications are to be avoided. All catheter care is done under sterile conditions with the patient in the modified Trendelenburg position (supine, head and neck-dependent, feet elevated) to minimize the risks of infection and air embolism.

One modification of the subclavian insertion technique has the advantage of removing the long needle used for subclavian venipuncture from the field once catheter placement is complete (Fig. 2). This modification, designed specifically for TPN use, requires the use of a 3 inch long 14 gauge needle, a 9-inch long, 16 gauge radiopaque silicone infusion catheter, an 18 gauge tubing adapter, and a 1-inch Teflon sleeve. After the infusion catheter is threaded through the needle and into the central venous system, the needle is carefully withdrawn over the catheter and from the field. An intravenous infusion with an in-line, 0.45 μ filter is then connected to the infusion catheter using an 18 gauge adapter. A small Teflon sleeve protects the catheter-adapter junction and provides a point of stability for securing adapter, catheter, and sleeve to the anterior chest wall with nonabsorbable suture (see Fig. 2). A second suture, located at the catheter exit site through the skin, is

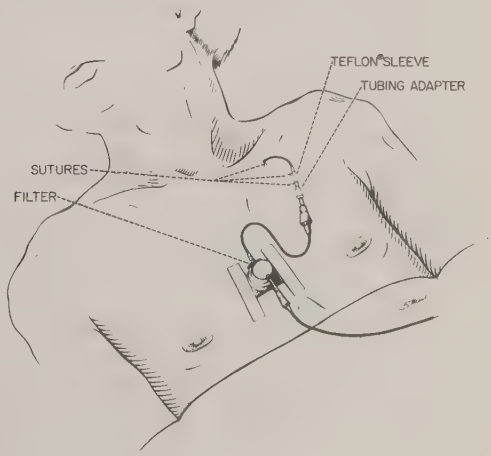

Figure 2. Detail of subclavian catheter skin exit site.

secured to the catheter tight enough to prevent motion at the skin exit site. An iodophor antibiotic ointment is used to seal the skin exit site, after which a sterile dressing which seals the exit site, external catheter, and adapter-filter junction within the dressing is applied. Mask and gloves are used whenever this dressing requires changing. No TPN solution is infused until a chest film demonstrates the tip of the infusion catheter to be within the superior vena cava.

CARE OF THE INTRAVENOUS FEEDING CATHETER

Once placed with its tip into the superior vena cava, the intravenous feeding catheter is used for no other purpose than to administer TPN therapy. The intravascular silicone catheter is not changed for the duration of therapy unless catheter sepsis, venous thrombosis, or displacement of the intracaval catheter tip occurs. The catheter dressing and terminal filter are changed routinely every 48 hours by members of the TPN team. Nonscheduled changes are performed only by trained physicians or nursing personnel. All dressing changes require that the patient be masked and placed in the modified Trendelenburg position. Using mask and gloves, a member of the TPN team (1) removes the old dressing, (2) observes the condition of the skin at the catheter exit site, (3) cleans the skin with an organic solvent, (4) paints the exit site and adjacent chest wall with an iodophor solution, (5) applies iodophor ointment to the exit site, (6) changes the terminal filter, and (7) applies a new, sterile, sealed gauze dressing. The terminal 0.45 μ filter serves as an air, particulate matter, and partial microbial barrier.

The bottle of appropriate TPN solution is administered to the patient through an intravenous tubing which has a sterile, in-line volume measuring reservoir with an outlet tubing connecting to the 0.45 μ filter (Fig. 3). Proper nursing attention to the flow of solution through the volume reservoir obviates the need for an infusion pump in most nursing units. Prescribed hourly flow rates must be met if the risks of hypoglycemia, hyperglycemia, and fluid overload are to be avoided. The intravenous line and volume reservoir are changed with each new bottle of TPN solution.

PRIORITIES OF THERAPY

Providing an extracellular environment conducive to substrate utilization is of prime importance any time nutrients are given. In general, patient vital sign stability, hydration, and electrolyte balance are prerequisite to either the initiation or acceleration of TPN therapy. More specifically, patients in crisis situations such as those related to an inadequate airway or respiratory function, an improper blood volume (dehydration, hemorrhage, shock, congestive failure), sepsis (bacteremia or fungemia), electrolyte or mineral imbalance, or hypoglycemia have an extracellular environment which may interfere with normal cellular transport mechanisms; the therapy of these emergencies must receive

temporary priority over nutrient considerations until metabolic stability has been achieved. The importance of these general principles cannot be overemphasized.

Following stabilization of the crisis situation, TPN may be instituted. Patients suffering from the stress of multiple trauma, burns, or infection are hypermetabolic and soon become candidates for TPN. Stress produces an endogenous hyperglycemia secondary to glycogenolysis and gluconeogenesis. During stress the patient may temporarily require exogenous supplementation of insulin in order to maintain glucose homeostasis and to avoid glucosuria. If insulin is needed to prevent hyperglycemia with glucosuria, sufficient insulin should be given. As stress abates, the insulin requirement will rapidly diminish and care must be taken to avoid hypoglycemia. Regular insulin may be added directly to the nutrient solution or given according to a sliding scale.[33]

THE CHOICE OF SOLUTIONS

The general indication for TPN is gastrointestinal failure, and most solutions available today are designed specifically for this problem. Pa-

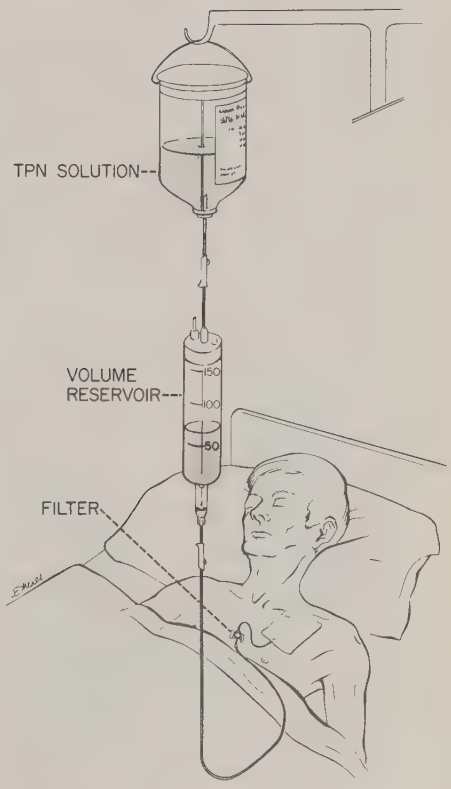

Figure 3. Bedside TPN delivery system.

Table 2. *Base Intravenous Nutrition Solutions*

COMPOSITION PER LITER	SOLUTION A	SOLUTION B
Amino acids	500 ml Travasol 5.5 per cent*	500 ml FreAmine II 8.5 per cent†
Dextrose	+500 ml 50 per cent dextrose monohydrate	+500 ml 50 per cent dextrose monohydrate
	1000 ml total volume	1000 ml totál volume
Approximate carbohydrate calories	850	850
Total nitrogen (gm per liter)	4.63	6.25
Carbohydrate calories per gram nitrogen	180	135
Electrolytes (mEq per liter)		
Potassium	30	(20)‡
Phosphate	30	10
Magnesium	5	(5)‡
Sodium	35	5
Chloride	35	(20)‡
Acetate	50	–

*Travasol (amino acid) injection with electrolytes, Travenol Laboratories, Inc., Deerfield, Illinois.
†FreAmine II (amino acid injection), McGaw Laboratories, Irvine, California.
‡As additives (20 mEq KCl and 5 mEq $MgSO_4$).

tients with gastrointestinal failure and concurrent renal or hepatic failure require solution modification and thus must be considered separately.

Gastrointestinal Failure

Two solutions which are generally available and designed for the treatment of gastrointestinal failure are indicated in Table 2. Assuming sufficient volume is given via a central venous route, both solutions can meet carbohydrate and amino acid needs for an indefinite period of time. As therapy continues, individual patient needs for water, vitamins, electrolytes, minerals, essential fatty acids, and trace elements must be met by either parenteral or enteric routes. Central venous administration is *required* of only the carbohydrate-amino acid mixture.

Both solutions in Table 2 are crystalline amino acid solutions which provide: (1) approximately 0.85 carbohydrate calorie per milliliter, (2) nitrogen in the form of readily utilized L-amino acids in ratios well tolerated in humans, (3) more than the minimal daily requirement of essential amino acids in each liter,[44] and (4) a carbohydrate calorie to gram nitrogen ratio of 135 or above. Protein hydrolysate mixtures are not included in our present solution formulation.

The two solutions differ both in amino acid concentrations and in concentrations of electrolytes, especially potassium and magnesium. Potassium, phosphate, and magnesium are the three major intracellular ions (Fig. 4). The need for these ions becomes increased as positive nitrogen balance, protein synthesis, intracellular shift of nutrient, and cellular division proceed. To provide for these needs, 20 mEq KCl and 5 mEq $MgSO_4$ are routinely added to each liter of solution B (as indicated) to meet intracellular requirements in patients with normal renal function.

These solution recommendations should not be interpreted to mean that there is a "standard" formula which will meet the needs of all pa-

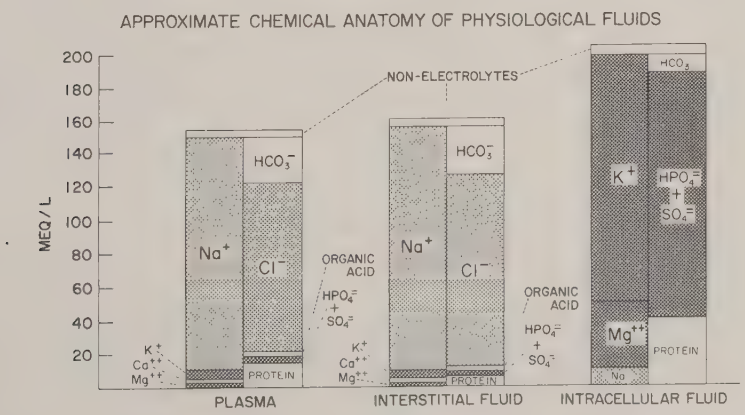

Figure 4. Chemical composition of plasma, interstitial fluid, and intracellular fluid. (Modified from Gamble, J. L.: Chemical Anatomy, Physiology, and Pathology of Extracellular Fluid. Cambridge, Mass., Harvard University Press, 1958.)

tients. Good starting formulas, however, are essential to good therapy. By increasing or decreasing the concentration of each electrolyte as indicated, the solution then becomes individualized.

In addition to the special intracellular needs indicated, total patient needs may include major amounts of water, sodium, potassium, chloride, and bicarbonate resulting from such extracellular losses as open wounds, fistulas, nasogastric losses, diarrhea, or high output renal failure. Replacing these fluid losses is essential; collection and measurement of both volume and electrolyte content are excellent guides to replacement. Impending electrolyte imbalance can often be predicted by observing trends in the metabolic flow sheet; simultaneous serum and urine electrolyte determinations provide an index as to whether the kidneys are responding by conserving or excreting a particular electrolyte. Initiating TPN in these patients may require titrating the extracellular needs (peripheral venous route) with the nutritional and intracellular needs (central venous route) until the total fluid and electrolyte requirement can be supplied safely within the volume constraints of the TPN solution.

Both the estimated human daily vitamin requirement and the usual routes of vitamin administration during TPN are indicated in Table 3.[23, 41] Calcium must be included in the total nutritional plan. If included

Table 3. *Recommended Adult Male Vitamin Allowance*

VITAMIN	RECOMMENDED DAILY ALLOWANCE[41]	USUAL ROUTE AND DOSAGE DURING TPN*
A Activity	5000 IU	10,000 IU
B Complex		
Thiamine (B_1)	1.4 mg	50 mg
Riboflavin (B_2)	1.6 mg	10 mg
Niacin	18 mg	100 mg
Vitamin B_6	2.0 mg	15 mg
Folacin	400 μg	5 mg intramuscularly both initially and weekly
Vitamin B_{12}	3.0 μg	500 μg intramuscularly both initially and monthly
Ascorbic Acid	45 mg	500 mg
D	400 IU	1000 IU
E Activity	15 IU	5 IU
K	Not established	10 mg intramuscularly initially and weekly

*Vitamins are routinely administered as one ampule of M.V.I. which is added to only 1 liter of intravenous fluid each day. Exceptions are folic acid, vitamin B_{12}, and vitamin K as noted.

M.V.I. (Multi-Vitamin Infusion), USV Pharmaceutical Corp., Tuckahoe, N.Y.

in the TPN solution, calcium precipitates must be avoided. A second approach includes the intermittent administration of calcium using either peripheral intravenous or enteric routes.[10, 23, 34, 48] Regular insulin, when needed in diabetic patients, is added directly to the TPN solution in dosages of 15 to 20 units regular insulin per liter of solution. Supplemental subcutaneous insulin can also be given if necessary.[22, 23]

Renal Failure

Patients with combined gastrointestinal and renal failure (creatinine clearance <25 ml per min) require a solution composition very different from that previously described. Volume restrictions in the renal failure patient require that a base nutritional solution have (1) a high caloric to nitrogen ratio, (2) a high proportion of essential amino acids, and (3) minimal concentrations of electrolytes and minerals in order to meet the unique needs of the patient with little or no urinary output.[1, 2, 20] Patients receiving these solutions usually require extensive metabolic monitoring. None of these special solutions are presently available for general use, though release of such solutions may be imminent.

Hepatic Failure

Patients with combined gastrointestinal and hepatic failure present a special dilemma since residual liver function and encephalopathy may be especially vulnerable to the amino acid profile of the TPN solution. Patients with minimal, chronic, alcoholic cirrhosis may tolerate either of the solutions indicated in Table 2 without ill effect; certainly frequent monitoring for possible adverse trends of serum SGOT, SGPT, alkaline phosphatase, bilirubin, BSP, and ammonia is mandatory in these patients. Worsening liver function would indicate a slowing or discontinuance of the solution. Diagnostic interpretations of serum enzyme studies are complicated by the fact that a frequent temporary response to TPN in patients with no known liver disease is an elevation of serum SGOT and alkaline phosphatase.[26, 31]

Patients with advanced liver disease, hepatic encephalopathy, or hepatic coma who require parenteral nutrition present a multitude of therapeutic problems, most of which are being pursued by several investigative teams.[24, 25, 31, 38] Considerable investigative work remains before a parenteral nutrition solution will be available for general use in patients with this degree of liver failure.

INITIATING PARENTERAL NUTRITION

The decision to initiate TPN therapy may carry with it many medical, ethical, and socioeconomic implications, as the therapy of many of these patients will need to be both precise and prolonged. If the responsible physician cannot give affirmative answers to the following questions, then physician, hospital, or patient preparedness for TPN is probably lacking:

1. Does the patient have potential for recovery?
2. Is *parenteral* nutrition required?
3. Has informed consent been obtained?
4. Has adequate nursing and TPN team support been made available?
5. Is the tip of the intravenous feeding catheter within the superior vena cava?
6. Has the most appropriate solution been selected?
7. Has the metabolic flow sheet been initiated and have pre-TPN data been entered?
8. Does the patient have hemodynamic stability, adequate hydration, and normal serum electrolytes?

Minimal baseline metabolic data include patient weight and regular (at least every 4 hours) temperature, pulse, respiration, and blood pressure, as well as total intake and output for the 24-hour interval immediately preceding TPN infusion. Also necessary are pre-TPN values for complete blood count, serum glucose, urea, sodium, potassium, bicarbonate, chloride, osmolarity, phosphate, magnesium, calcium, albumin, total protein, bilirubin, alkaline phosphatase, PTT, and SGOT. In patients with multiple sources of extracellular fluid loss, daily or frequent collections (both prior to and during TPN) of 24-hour urine, wound, fistula, or diarrhea specimens with subsequent determinations of volume, sodium, and potassium losses may be necessary for accurate fluid and electrolyte replacement. In patients with normal renal function, frequent monitoring of urinary osmolarity may serve as an index to degree of patient hydration.

Frequent evaluation of the patient for signs of fluid overload is an essential component of all TPN techniques and is especially important during the first few days of therapy. As patient monitoring proceeds, central venous feeding is initiated at an appropriate rate (usually 50 ml per hr) for the first 24-hour interval. Assuming no adverse trends in patient general status, vital signs, serum glucose, BUN, or electrolytes, the TPN flow rate is increased by increments of 25 ml per hr each day as peripheral intravenous fluids are tapered off until fluid and electrolyte balance at an optimal level of caloric intake is achieved.

Most adults of average size who are not hypermetabolic do well with intravenous carbohydrate caloric intakes of 3000 to 3500 calories per day. Higher caloric intakes generally require monitoring of serum BUN, glucose, and electrolytes at more frequent than daily intervals. Serum phosphate, calcium, total protein, albumin, bilirubin, alkaline phosphatase, SGOT, PTT, and osmolarity are measured every 4 to 5 days or more frequently if needed.

Once initiated, TPN solutions cannot be discontinued abruptly without exposing the patient to the risk of rebound hypoglycemia.[19] If the central venous feeding infusion should stop for any reason, an infusion of 10 per cent glucose should be promptly initiated and maintained for a 24-hour interval or until central venous feedings resume. At the termination of TPN therapy, all patients are slowly tapered off the concentrated dextrose solution by substituting 10 per cent dextrose in water for the TPN solution and infusing the dextrose solution at a progressively slower rate during the subsequent 24-hour interval.

COMMON METABOLIC AND NUTRIENT PROBLEMS

Metabolic problems to be anticipated at all stages of TPN therapy using the carbohydrate-amino acid solutions described are indicated in Table 4. While not inclusive of all the metabolic situations that may arise in critically ill patients, those problems are described which are solution-related and which require prompt correction before safe and effective therapy can be expected. Problems unique to pediatric patients or to the use of protein hydrolysates, unbalanced amino acid solutions, or intravenous fats are not included in Table 4.[14, 19, 42, 51, 52]

In addition to the common metabolic problems described, more subtle deficiency problems must be anticipated and prevented if TPN is to be effective. The two solutions described are designed to meet the usual daily carbohydrate and amino acid needs in patients with good

Table 4. *Common Metabolic Complications of TPN Encountered in Adults*

COMPLICATION	POSSIBLE ETIOLOGY
I. Carbohydrate Metabolism	
A. Hyperglycemia	1. Excessive rate of infusion of glucose 2. Insufficient endogenous insulin secretion 3. Sepsis 4. Glucocorticoids
B. Hyperosmolar nonketotic dehydration (hyperosmolar syndrome)	1. Persistent hyperglycemia 2. Osmotic diuresis 3. Dehydration
C. Hypoglycemia	1. Abrupt interruption of TPN infusion 2. Excessive insulin
II. Amino Acid Metabolism	
A. Elevated blood urea nitrogen	1. Intrinsic renal disease 2. Dehydration 3. Excessive rate of infusion of amino acids 4. Low caloric:nitrogen ratio of TPN solution
B. Hyperammonemia	Intrinsic liver disease
III. Electrolyte and Mineral Metabolism	
A. Hypokalemia	Insufficient potassium intake relative to losses and anabolic requirements.
B. Hypophosphatemia	Insufficient phosphate intake relative to losses and anabolic requirements.
C. Hypomagnesemia	Insufficient magnesium intake relative to losses and anabolic requirements.
IV. Hematologic	
A. Anemia	Iron, folic acid, vitamin B_{12}, or copper deficiency
B. Bleeding diathesis	Vitamin K deficiency

Table 5. *Nutritional Deficiencies During Prolonged TPN*

DEFICIENCY	SUPPLEMENTAL FEEDING TECHNIQUE
Water	1. Decrease rate of TPN infusion. 2. Administer 5 per cent dextrose in water intravenously. 3. Enteric feedings; water or dilute nutritional solution. 4. Humidify inspired air.
Iron	Appropriate dosage of intramuscular iron preparation.
Trace elements (copper, zinc, manganese, chromium, cobalt, fluoride.)	1. Present in variable and usually insufficient amounts as contaminants in TPN solutions; present to some degree in blood and blood products. 2. Enteric feedings; trace element concentrates, elemental diets, liquid or higher diets.
Essential fatty acids	Replace using enteric feedings, intravenous fat preparations, or percutaneous techniques.[3, 30, 40, 43]

renal and hepatic function. While meeting this objective, the solutions do not meet the highly variable individual patient needs for water, electrolytes, minerals, vitamins, trace metals, and essential fatty acids. Therapy can often be simplified by bypassing the central venous route when administration of nonhyperosmolar nutrients is indicated. Various supplemental techniques commonly used to prevent or treat nonsubstrate nutritional deficiencies are indicated in Table 5. While not always utilized during the first few days of TPN therapy, these techniques become of increasing importance as therapy is prolonged.

EVALUATING THE FEBRILE PATIENT

Any temperature in a patient receiving TPN requires prompt evaluation and therapy if the risk of intravascular seeding of organisms either from or to the indwelling feeding catheter is to be minimized. Initial patient evaluation should include diagnostic investigation and subsequent treatment of common sources of fever (skin lesions, wounds, abscesses, lungs, urinary tract, phlebitis) prior to ascribing all symptoms to the feeding catheter.

In the febrile patient where a source of infection other than the central venous catheter is suspect, catheter evaluations in situ can include a dressing change with inspection and culture of the skin exit site, blood cultures drawn through the catheter itself using aseptic technique, and culture and replacement of the TPN solution, volume reservoir, intravenous tubing, and terminal filter. Indications for prompt removal of a catheter temporarily left in situ would include a lack of patient defervescence, inflammation at the skin exit site, a positive catheter-drawn blood culture, or continued suspicion of catheter infection. Negative catheter-drawn blood cultures can mean either that catheter sterility is present or that organisms present are not being grown on culture media.

Unexplained fever or sepsis implicates the central venous catheter and prompt, definitive management is indicated. A new intravenous line is placed, 10 per cent dextrose is infused, and aseptic removal with culture of the subclavian catheter tip is performed. Defervescence within 48 hours implicates the catheter as being the source of the fever, a diagnosis often confirmed by catheter culture studies. Catheter studies must always include cultures for aerobic and anaerobic bacteria as well as fungi. Once the central venous catheter is removed, a new one is not reinserted until all evidence of bacteremia and fungemia is gone. Continuous carbohydrate support with a peripheral infusion of 10 per cent dextrose is maintained until adequate nutrition can be provided.

PARENTERAL NUTRITION, ANESTHESIA, AND SURGERY

The risk of intraoperative hypoglycemia is a special consideration in the patient receiving TPN who is about to undergo a surgical procedure. This potential complication must be prevented, and maintaining a continuous glucose infusion during the entire operative interval has been effective. Either of two techniques is generally used. The first technique consists of substituting 10 per cent dextrose in water for the TPN solution on the day of surgery and postoperatively, maintaining a rate of solution infusion which is comparable to the presurgical flow rate. A second technique consists of using the TPN solution (25 per cent dextrose, amino acids, and electrolytes) during the operative interval at a rate of infusion which is approximately half the usual presurgical flow rate. Both techniques prevent intraoperative hypoglycemia without producing complicating hyperglycemia, though monitoring of serum glucose levels during the operative interval is indicated. The second technique may be advantageous in that sufficient glucose is provided in a smaller volume, reducing the risk of total fluid overload as the patient's requirements for glucose, fluids, blood, and electrolytes are met both during and after surgery. Parenteral nutrition resumes its presurgical priority after fluid, blood, and electrolyte requirements are met during the first few postoperative days.

In addition to the potential problems of glucose metabolism described, patients on TPN requiring major surgery may be especially vulnerable to the TPN hazards of hypokalemia, hypophosphatemia, and hypomagnesemia, especially during the first week of TPN therapy. If emergent surgery and general anesthesia are required early in the course of TPN therapy, special attention must always be directed toward maintaining normal serum glucose, electrolyte, phosphate, and magnesium levels during the operative interval.

RESULTS OF THERAPY

Using the principles and techniques of TPN therapy described, the experience within the Department of Surgery at Loyola University Medical Center from January 1, 1973, to January 1, 1976, has been reviewed.

Table 6. *Primary Diagnoses in 73 Patients Requiring Parenteral Nutrition*

PRIMARY DIAGNOSIS	NUMBER OF PATIENTS
Gastrointestinal cancer	18
Enterocutaneous fistula	13
Peptic ulcer disease	8
Inflammatory bowel disease	8
Pancreatitis	6
Intra-abdominal abscess	5
Ischemic bowel disease	4
Esophageal stricture	3
Other	8
TOTAL	73

During this 3-year interval there were 73 patients who received 1993 days of therapy with carbohydrate-crystalline amino acid solutions. All patients were monitored daily by members of the TPN team. Forty-two patients received solution "A" (see Table 2); 31 patients received solution "B". Vitamins, electrolytes, and minerals were provided as necessary. Intravenous fat solutions were not used. Efficacy of therapy was demonstrated by the performance of daily nitrogen balance studies in 18 patients.

Patients selected for therapy were those who were seriously ill, had total gastrointestinal failure for a minimum of 7 days, and had no prospect of return of gastrointestinal function in the foreseeable future. All patients treated were adults; 62 were males. The average age was 44 years with a range from 17 to 76 years. The average duration of in-hospital TPN therapy was over 27 days, reflecting the fact that many of these patients were transferred from other hospitals specifically for TPN therapy following multiple previous surgical procedures. Primary diagnoses for these patients are indicated in Table 6. Patients with a primary diagnosis of cancer were thought to have controlled tumor but developed postsurgical complications requiring TPN therapy. Fifty-three patients (73 per cent) survived their illness and were discharged from the hospital, though two of this group required the continuation of TPN as outpatients. No deaths were attributable to TPN therapy.

Complications of TPN therapy encountered in these 73 patients have been divided into two groups, catheter-related and metabolic, and both groups are listed in Table 7. Subclavian thrombosis was diagnosed in three nonseptic patients who abruptly developed edema of the ipsilateral face, neck, and supraclavicular area without evidence of catheter tip displacement or catheter infection. All three patients were treated by removal of the subclavian catheter with subsequent disappearance of edema within 72 hours. Both patients with catheter sepsis responded promptly to removal of the catheter and systemic antibiotics. Each suspect catheter grew organisms—one Staphylococcus aureus, the second Candida albicans. The patient who developed a pneumothorax during subclavian catheterization responded to tube thoracostomy.

Table 7. *Complications Encountered in 73 Patients Requiring Parenteral Nutrition*

COMPLICATION	NUMBER OF PATIENTS
I. Catheter Related	
1. Subclavian thrombosis	3
2. Catheter sepsis	2
3. Pneumothorax	1
II. Metabolic	
1. Hyperglycemia	Frequently encountered in mild degrees, corrected as TPN continued.
2. Hypokalemia	
3. Hypophosphatemia	
4. Elevation of serum urea	
5. Hypoglycemia	2
6. Hyperosmolar syndrome	2
7. Copper deficiency	1
8. Zinc deficiency	1
9. Essential fatty acid deficiency	1

Metabolic complications encountered were numerous, most were nonthreatening, and all were difficult to ascribe entirely to TPN in this seriously-ill group of patients. Mild degrees of hyperglycemia, hypokalemia, hypophosphatemia, and elevation of serum urea levels were frequently encountered and corrected as therapy proceeded. Hyperglycemia (serum glucose >180 mg per dl) was corrected by adjusting the rate or constancy of the TPN infusion or by providing insulin for patients with diabetes. Hypokalemia and hypophosphatemia were corrected with intravenous supplements of potassium or phosphate. Elevated serum urea levels (in the absence of intrinsic renal failure) were treated by decreasing the rate of TPN infusion and/or increasing the level of patient hydration.

Two patients developed "rebound" hypoglycemic episodes; both were treated with intravenous glucose. The hyperosmolar syndrome (patient disorientation or lethargy, intense thirst, osmotic diuresis, serum osmolarity between 310 and 318 milliosmoles per liter) was diagnosed in two patients, both of whom responded to reducing the rate of TPN infusion and supplementing the total water intake with intravenous 5 per cent dextrose in water.

Essential fatty acid, zinc, and copper deficiencies were each diagnosed once near the end of prolonged therapy in separate patients. The EFA-deficiency responded to combined percutaneous and enteric therapy. The zinc and copper deficiencies responded to enteric feedings which contained zinc and copper.

SUMMARY

Total parenteral nutrition has evolved as a distinct therapeutic reality within the past decade. Starvation or malnutrition need no longer be

accepted as a necessary component of prolonged illness. Though current TPN techniques can be both safe and effective, the prevention of potential complications must always have a high priority. Changes in technique are to be anticipated as further knowledge and improved materials allow the pursuit of more basic clinical problems.

The recent experience with the use of high caloric TPN solutions for prolonged gastrointestinal failure in 73 patients at the Loyola University Medical Center has been summarized. The need for the involvement of an experienced TPN team in the care of these patients cannot be overemphasized if the numerous and diverse potential complications of the TPN system are to be minimized.

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Surgery of the Vermiform Appendix

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Though this section is being devoted to the complications of surgery of the vermiform appendix itself, it is fair to say that an unnecessary operation is in itself a complication which is to be avoided whenever possible. The incidence of negative surgical exploration for acute appendicitis varies from 18 to 42 per cent in most large series (Table 1). One series reports a diagnostic error of 45 per cent in women between the ages of 20 and 40.[29]

Classical teaching specifically contraindicates the performance of a barium enema in the face of acute appendiceal disease, primarily because of the fear of leakage of barium in the presence of appendiceal perforation. In direct contrast to the above edict, the barium enema has been used frequently at our institution in the diagnosis of acute appendicitis with absolute safety and a marked reduction in the incidence of negative exploration.

Appendiceal luminal obstruction is usually a prerequisite for the occurrence of acute appendicitis. Therefore, radiologic demonstration of a patent appendiceal lumen should rule out appendicitis in most cases. In addition, and even more important, the mesoappendix and adjacent omentum often become edematous and produce deformity of the cecum in appendicitis. This manifests itself as an indentation adjacent to the origin of the appendix at the cecal base. The performance of postevacuation and delayed films will significantly increase the yield of visualization of the appendix.[48] The radiologic findings suggestive of acute appendicitis are (1) persistent nonvisualization of the appendix, (2) partial visualization of the appendix, (3) pressure defects on the cecum, and (4) irritability of the cecum and terminal ileum (Fig. 1). Nonvisualization of the appendix by itself is not diagnostic of acute appendicitis, since 5 to 10 per cent of normal appendices will not visualize.[48] This may be due to many factors such as fibrous obliteration of the lumen, obstruc-

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Table 1. *Incidence of Negative Surgical Exploration in Acute Appendicitis*

HOSPITAL	CASES	DIAGNOSTIC ERROR
Massachusetts General Hospital[5]	5800	18 per cent
Burbank Hospital[47]	2322	42 per cent
San Francisco General Hospital[29]	1000	20 per cent
Females 20 to 40		45 per cent
Western Infirmary (Glasgow)[26]	460	
Surg. A		22 per cent
Surg. B		24 per cent
Surg. C		31 per cent
Surg. D		30 per cent
Surg. E		33 per cent

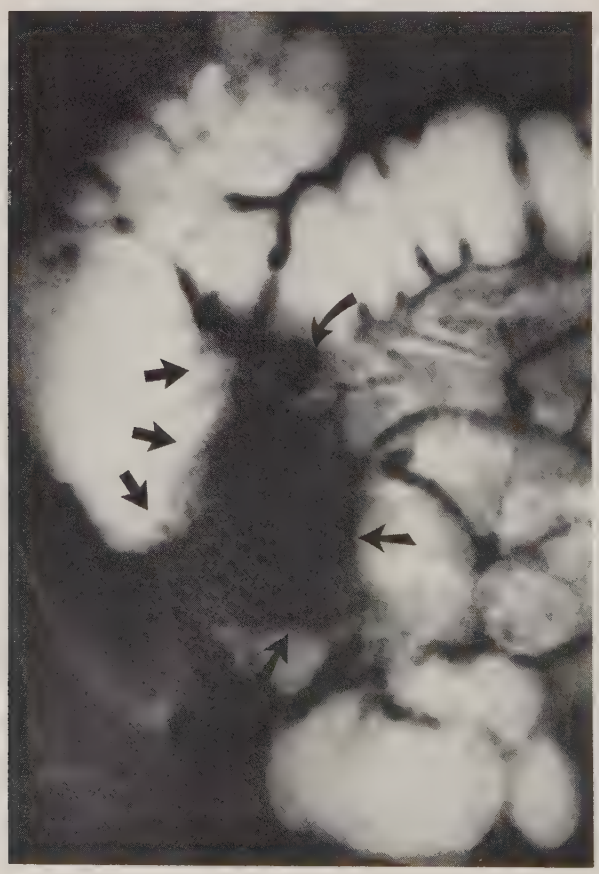

Figure 1. Radiologic findings suggestive of acute appendicitis.

tion by a fecolith without concomitant appendicitis, or congenital failure of canalization.

During the years 1973 to 1975, 489 cases were admitted to our hospital with suspected appendicitis. In this group, 218 barium enema examinations were carried out. The studies were performed with an unprepared colon, and were often completed before the CBC was reported. The detailed experience in this group has been reported elsewhere, but will be summarized here.[45]

Each year of the study showed a decreasing incidence of operation for appendicitis which was felt to be due in large part to more accurate diagnosis. Also, the number of barium enemas performed each year for appendicitis rose steadily, which was felt to represent an increasing acceptance of the test as a useful, safe, diagnostic tool. Ninety-seven per cent of those cases diagnosed radiologically as acute appendicitis were confirmed by operation. The incidence of radiologically negative cases operated upon because of compelling clinical indications and found to have appendicitis was 7 per cent. It is important to emphasize at this point that in all cases the complete clinical picture must determine whether operation is indicated. The barium enema has proven to be a useful adjunct in a patient with a complicated clinical presentation, but should not be relied upon completely to the exclusion of standard methods of evaluation. The majority of the patients with suspected appendicitis found to have a normal lower GI series were females between the ages of 11 and 40. It is in this group that we feel the barium enema plays its most important role. A significant proportion of this group would probably have undergone exploration on clinical grounds since females in the menstruating age group traditionally represent the greatest diagnostic challenge in appendicitis.

Barium enema was performed only when a diagnostic dilemma existed and infrequently in males. It has lowered our negative exploratory rate to 9.15 per cent. An unexpected occasional bonus from this procedure has also been the detection of an incompletely rotated cecum allowing for more accurate planning of the incision.

THE INCISION

The incision of choice in the patient in which the diagnosis of appendicitis is felt to be very secure is the McBurny incision (McBurny-McArthur) or its modification – the Rockey-Davis incision. The Fowler-Wier modification adds the incision of the anterior rectus sheath with medial retraction of the rectus muscle and incision of the posterior rectus sheath.[3] Although many surgeons have been trained to use a right paramedian incision whenever the patient is in the menstruating age group, we would reserve this approach for those cases in which there are some atypical features in the presentation. Whenever any one of the right lower quadrant muscle-splitting incisions is used, it should be located higher than traditionally described, especially if a transverse skin incision is used. If the appendix is lower than the incision, it is a simple matter to mobilize the cecum superiorly into the wound. On the

other hand, if the incision is placed low, it may be impossible to mobilize the cecum and appendix inferiorly. If the cecum cannot be located in the right lower quadrant or if the findings indicate a process which cannot be readily managed via an established muscle-splitting incision or one of its modifications, a second vertical incision is strongly recommended. To struggle with the existing incision and poor exposure will often violate one of the basic axioms of surgery, namely, good exposure, and lead to unnecessary complications.

If the appendix is normal and one is operating through a vertical incision, there is no problem since a thorough systematic exploration can be undertaken to rule out all causes of the acute surgical abdomen. The problem arises when the appendix is normal and the operation is being carried out through a right lower quadrant muscle-splitting incision. Once it is determined the appendix is normal, evaluation of both ovaries and uterus is carried out prior to appendectomy. In the thin patient, they can be seen and often prolapsed into the field. This may require extending the incision with division of the rectus sheaths and medial retraction of the rectus or division of the rectus, especially in the more obese patient.[3] If these structures are normal, the terminal ileum and its mesentery are examined for a distance of about 2 feet to rule out regional enteritis, a Meckel's diverticulum, and mesenteric adenitis. If examination of these structures has failed to elucidate the diagnosis and there is no evidence of peritonitis, peritoneal contamination, or blood, an appendectomy should be carried out. At this point the question which arises is how convinced are you that you are dealing with a surgical abdomen. The answer to this question will help to determine how far further investigation of the abdomen should go. Obviously, if there is evidence of peritonitis or blood in the abdomen, a satisfactory explanation must be found even if an additional vertical incision is required.

In one large series of patients (820) in which the preoperative diagnosis was acute appendicitis and who proved to have normal appendices and no other surgical disease, mesenteric adenitis accounted for the symptoms leading to operation in 36 per cent, ovarian cyst rupture without significant hemorrhage in 14 per cent, and no apparent lesion in 50 per cent.[23]

INCISIONAL COMPLICATIONS

The most common incisional complication encountered is infection. The incidence of wound infection in primarily closed wounds correlates well with the degree of appendiceal inflammation. In an excellent study of 539 appendectomies, it was found that the incidence was 0.3 per cent in negative appendices, 7.4 per cent in the acute purulent cases, 16.4 per cent in the perforated cases with localized peritonitis, and 35 per cent in the perforated patients with generalized peritonitis.[28] Thus, earlier diagnosis with reduction in the percentage of perforated cases is the initial step towards reduction of wound infection.

The type of incision made is a factor, as well as how the incision is made. McBurny incisions usually have lower wound infection rates than vertical incisions and thus are preferable if possible.[12] Minimal undermining of the subcutaneous tissues is essential to avoid creation of dead space. This can be accomplished easily with an oblique incision; however, a transverse incision can also be used with simple retraction of the subcutaneous tissue rather than undermining. We feel that inversion of the appendiceal stump, though controversial, should be carried out where feasible unless there is severe edema of the cecal wall. This acts to bury the mucosal surface of the appendix and reduces the opportunity for contamination of the peritoneum and the wound.

Drainage of the peritoneal cavity is reserved for specific indications since its use has been demonstrated to increase wound infection rates.[33] When indicated (example localized abscess) drainage should be carried out through a separate stab wound to minimize wound contamination.

Closure of the incision should be carried out with either a monofilament nonabsorbable suture or chromic catgut. Multifilament sutures in contaminated wounds are associated with higher infection rates and should be avoided.[2] The newer synthetic absorbable sutures such as polyglycolic acid may also prove to be acceptable, but a more complete trial is necessary. Closure of the subcutaneous fat is not only unnecessary, but is contraindicated since the suture will enhance bacterial growth. Despite the widespread use of antibiotics, there has actually been little concrete evidence to show that they reduce the incidence of wound infection in this disease.[28] It is hoped that greater use of antibiotics, begun in the preoperative period in cases where perforation is suspected, will eventually help reduce the infection rate. Use of a combination of two antibiotics (cephalothin and clindamycin) to cover both aerobic gram-negatives and the anaerobes is probably worth consideration in cases where the likelihood of perforations is high. If, at the time of operation, it is found that the appendicitis is early, the antibiotics can then be stopped. However, if there is gangrene, pus, or perforation, they are continued therapeutically for 3 to 5 days.[16]

Wound closure or nonclosure is the final decision the surgeon must make in his effort to reduce the rate of wound infection in this disease. If the appendix is negative, it is reasonable to carry out primary closure. This is done after irrigation of the subcutaneous tissue with saline to remove devitalized fat, debris, and bacteria. In purulent appendicitis, antibiotic wound irrigation may play a role and has demonstrated a reduction in infection rates.[20, 40] When perforation and gross contamination are present, either secondary or delayed primary closure is indicated. Even with these techniques there will be cases of wound sepsis. It is important to wait sufficient time for the contamination to reduce in these wounds, often requiring 5 days before closure can be carried out. If at 5 days the wound is still not clean and granulating, it can be left open and allowed to heal without closure. This will prevent infection in the secondary closure, and actually will lead to an acceptable scar. Thus, there can be a place for primary closure, secondary closure, delayed

primary closure, or no closure at all. We consider this rational approach utilizing different methods preferable to a fixed approach such as always closing the wound or always leaving it open. Reasonably low wound infection rates can be obtained by carefully selecting the proper type of closure based on the degree of contamination.

INCIDENTAL APPENDECTOMY

"Incidental" removal of an easily accessible appendix during the performance of an unrelated abdominal operation is a widely practiced procedure with minimal morbidity. In general, we are in agreement with this practice. In patients beyond the age of 60 years, however, we would not perform incidental appendectomy because of the very low expected incidence of appendicitis in the elderly.[31, 32] Additionally, we do not perform this procedure during operations for the placement of a prosthetic arterial graft. Some surgeons question this practice under any circumstances since it has been argued that there may be an increased incidence of carcinoma of the colon (as well as carcinoma in other organs) in patients who have undergone appendectomy.[9, 36] More recent statistical analyses, however, successfully refute that claim.[7, 25, 38] Others argue that upon transecting the base of the appendix, bacterial contamination of an otherwise sterile operative field will occur. Inversion of the entire appendix into the cecum after division of the mesenteric blood supply and oversewing of the base has been proposed as an alternative procedure to prevent contamination.[21, 30] The devascularized inverted appendix, it is suggested, will then slough into the colon. Occasionally, however, sufficient mural blood supply remains to prevent necrosis of the appendix. Hemorrhage and intussusception[11, 34] have been reported as complications of persistent inverted appendices. Additionally, barium enema done years later may demonstrate the inverted appendix as a mass lesion. Even with the knowledge that an appendiceal inversion had been done, it is usually impossible to absolutely differentiate an inverted appendix from a neoplasm. Open surgical exploration has been necessary in several reported cases to make this differentiation. Recently we encountered this situation when a 68-year-old female, who had undergone cholecystectomy and inversion of the appendix 20 years earlier, was found to have a polypoid cecal defect on barium enema (Fig. 2*A*). In order to be certain the x-ray finding was not caused by a neoplasm, we performed fiberoptic colonoscopy and removed the inverted appendix. As is clearly seen, the mucosa is on the external surface of the appendix (Fig. 2*B*). Thus, in this case, colonoscopic excision averted an exploratory laparotomy.

Because of the potential problems of appendiceal inversion, we do not endorse its routine use. A more recent variation of the inversion technique using ligation of the base[10] to insure slough may prove acceptable, but there is too little experience with it at this time to recommend general usage.

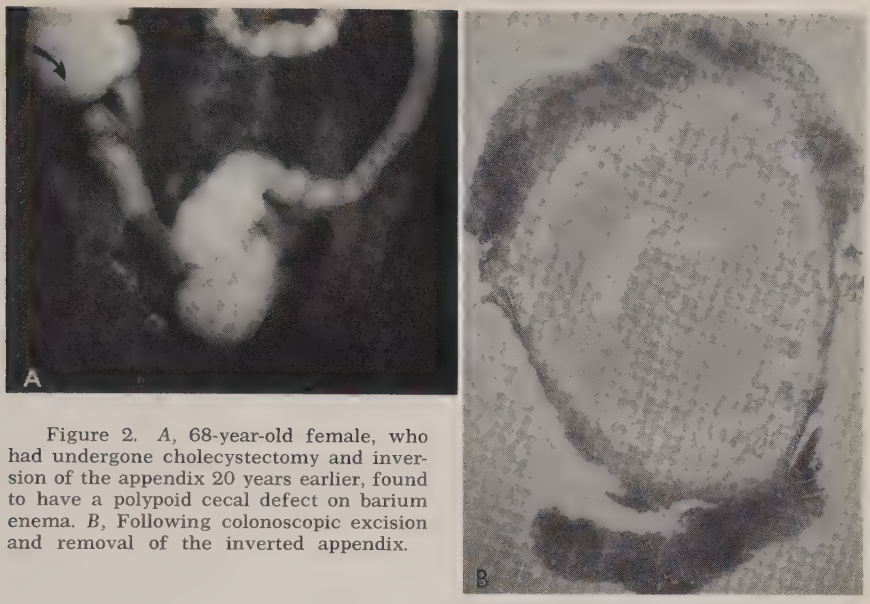

Figure 2. *A*, 68-year-old female, who had undergone cholecystectomy and inversion of the appendix 20 years earlier, found to have a polypoid cecal defect on barium enema. *B*, Following colonoscopic excision and removal of the inverted appendix.

APPENDECTOMY IN ASSOCIATION WITH INFLAMMATORY BOWEL DISEASE

It is well known that there is a high rate of fecal fistulae in patients with regional enteritis who undergo appendectomy. Colcock and Vansant reported a 25.6 per cent incidence in the cases from the Lahey Clinic in 1960.[14] This high rate of fistula has been substantiated by others who emphasize closing these patients without appendectomy.[13] Closer evaluation of the problem reveals that in this select group of patients with Crohn's disease proximal to the ileocecal valve, it is quite safe to carry out appendectomy. The apparent contradiction is resolved when one realizes that the fistulae that develop in these patients are usually from the area of the ileitis and not from the appendiceal stump. Both Ferguson and Crohn have reported no difficulty with fistula formation when appendectomy is carried out on these patients.[17, 18] An excellent review of this problem is presented by Marx who reviewed nine patients operated on for a presumed diagnosis of acute appendicitis and who were found to have regional ileitis.[35] Seven of these patients with uninvolved cecums underwent appendectomy without postoperative fistulae. An additional 12 patients were evaluated at their institution who had had appendectomy with associated Crohn's disease. Eight of these 12 cases had fistulae which were carefully evaluated by the surgeon and/or the pathologist. In none of these cases was the fistula demonstrated to come from the appendix, but rather was found to be associated with areas of ileum involved with Crohn's disease.

Based on this background, it is imperative to avoid appendectomy in patients with inflammatory disease involving the cecum or the appendix, but essential to carry out appendectomy in patients where the Crohn's disease exists proximal to the ileocecal valve. Appendectomy in this latter patient will avoid future confusion regarding the source of their abdominal pain. This is particularly important since it is well known that patients with regional ileitis can develop future appendicitis, which due to delay in diagnosis is often gangrenous or perforated.[14] Appendectomy at the initial exploration will avoid this potential future dilemma.

Cecal diverticulitis may also be confused with acute appendicitis. Diverticulosis confined to the cecum is not common, but has been reported as being the sole site in up to 2 per cent of cases.[6] When confined to the cecum, the diverticuli are frequently solitary and may well be congenital in origin. More commonly, however, right-sided diverticuli are found in association with diffuse acquired diverticulosis. The frequency of diverticulitis in the right colon is difficult to estimate though one series reports 1.2 per cent in cecum and 2.4 per cent in the ascending colon.[46] Cecal diverticulitis tends to occur at an earlier age (early 40's) than left-sided diverticulitis (mid-50's). The symptoms and findings of right-sided diverticulitis are very similar to appendicitis and in most cases a clinical diagnosis of appendicitis will be made. In those cases where the clinical picture is sufficiently ambiguous as to make the diagnosis of appendicitis questionable, a barium enema may be able to make a positive diagnosis. A history of previous appendectomy, of course, would make diverticulitis a more likely diagnosis.

If the clinical diagnosis of localized right colon diverticulitis can be made with reasonable certainty, conservative management is indicated. Progression of symptoms while on conservative management would demand immediate surgical exploration.

If a diagnosis of diverticulitis is made at the operating table and a solitary diverticulum is found with minimal surrounding inflammation, inversion of the diverticulum into the cecum with oversewing of the base has been found to be simple, effective, and safe. It there is too much local inflammatory induration of the diverticulum to permit inversion, then simple excision with a narrow rim of cecum and closure of the defect is indicated.[41] Appendectomy should be done at the same time.

The finding of a pericecal abscess due to diverticulitis but with no communication with the bowel lumen requires drainage only. If a large perforation with significant fecal spillage or a large abscess cavity is encountered, however, we believe drainage and a limited resection of the involved portion of the cecum and ascending colon with exteriorization of the distal colon and formation of a temporary ileostomy is indicated as the emergency procedure. Exteriorization of the perforation without resection may be preferred by some, but the after care of the large mass of right colon is more difficult and the dissection required is very little less than for resection. Diverticular perforation with minimal spillage of bowel content and limited inflammatory reaction may be safely handled by primary right colon resection and anastomosis.

A more difficult situation is encountered in those cases where no obvious diverticuli are present and the induration in the bowel wall cannot be distinguished easily from carcinoma. If, in the best judgment of the operator, the mass seems inflammatory and there is no other pressing reason for resection, such as bleeding or obstruction, then drainage and medical management are indicated. Follow-up barium enema after subsidence of the inflammation is then indicated for confirmation of the diagnosis. In cases where the likelihood of carcinoma is high, a right hemicolectomy is recommended despite the lack of proven carcinoma.[4, 19, 42]

CARCINOMA OF THE COLON AND APPENDICITIS

Acute appendicitis may also be associated with carcinoma of the colon. The frequency of this association is difficult to determine, but it is not very common. Many large series of acute appendicitis fail to mention a single case of associated carcinoma of the colon. Many series, however, do report the association, and in one series of 15 patients with appendicitis over the age of 50, three had carcinoma of the colon.[37]

The majority of carcinomas associated with appendicitis are located in the cecum,[22] though tumors more distal in the bowel are also reported.[37] The inflammatory reaction in the appendix may result from obstruction of its lumen by ingrowth of the cecal tumor or by spread of an inflammatory process from an adjacent ulcerated tumor. Indeed, the inflammatory process that leads to surgery may well be that surrounding the tumor, with the appendix being an innocent bystander.

It is more difficult to postulate how tumors more distal in the bowel might cause appendicitis. It is suggested that partial bowel obstruction with increased luminal pressure might interfere with normal drainage from the appendix. This along with a possible change in bacterial flora behind the partial obstruction may give rise to appendicitis. Raffensberger states that if acute appendicitis is encountered in infancy, there is a high likelihood that the patient also has Hirschsprung's disease.[44] The etiology here may also be luminal obstruction and bacterial overgrowth.

The preoperative diagnosis of a coexistent tumor and appendicitis is rare, but the presence of anemia, weight loss, or a mass in a patient over the age of 50 years should arouse suspicion. At the operating table, the tumor may well be missed. The mass of an adjacent cecal tumor may be interpreted as inflammatory induration from the appendicitis. Tumors more distal in the bowel may be missed because of reluctance to explore the abdomen in the face of appendicitis or because of limitations of the incision. For this reason, a follow-up barium enema should be done on all patients over the age of 45 years who have undergone appendectomy for appendicitis.

If a right colon tumor is recognized at the time of appendectomy and the local inflammatory reaction is not great, primary right hemicolectomy should be performed provided the general condition of the patient is satisfactory. Should an abscess be encountered, appendectomy

with drainage is the most prudent initial procedure with a plan to return at a later date for definitive resection. A free perforation of the tumor should be treated by right hemicolectomy if contamination is minimal or by resection with ileostomy and mucous fistula formation if generalized peritonitis is present.

Carcinomas of the left colon, if recognized at the initial procedure, are best treated by appendectomy with a plan for secondary resection on prepared bowel. If any hint of significant bowel obstruction is noted, however, decompression by either cecostomy or transverse colostomy should be done.

Occasionally a locally perforated carcinoma of the sigmoid colon will masquerade as appendicitis. Sigmoid resection with colostomy and a mucous fistula or a Hartmann procedure is the safest solution in this instance.

CARCINOMA OF THE APPENDIX

Adenocarcinoma of the appendix is a rare condition, reportedly present in from 0.3 to 0.8 per cent of removed appendices.[15] It is primarily a disease of the older age group with a maximum incidence in the sixth decade. The tumor, which is microscopically similar to adenocarcinoma of the colon, may be found at the tip of the appendix, but more frequently is located near the base. The tumor tends to spread via lymphatics, blood stream, and local extension much more frequently than does the more common carcinoid tumor.

There is no symptom complex or physical finding specific for this condition and the diagnosis will rarely be made preoperatively. More than one-half of reported cases have had a preoperative diagnosis of acute appendicitis. Luminal obstruction, tumor ulceration, or tumor perforation may initiate the inflammatory reaction. Because of the inflammatory induration, the tumor is frequently not recognized at the initial procedure. If no evidence of distant metastatic disease is present, right hemicolectomy is the treatment of choice. This may be done at the initial procedure if the diagnosis can be made. More frequently it will require a second operation since the diagnosis is often first suspected when the appendix is examined by the pathologist. Appendectomy alone is insufficient treatment for this disease. In a review of cases reported in the literature to 1973, it was found that, excluding cases with initial metastatic disease, appendectomy alone gave a 20 per cent 5-year survival, whereas right hemicolectomy gave 45 per cent.[24] Perforation of the tumor did not seem to affect survival rates.

MUCOCELE OF THE APPENDIX AND PSEUDOMYXOMA PERITONEI

Proximal obstruction of the lumen of the appendix associated with continued production of mucus distal to the obstruction may lead to distention and formation of a mucocele. Both benign and malignant varie-

ties occur. Unlike the adenocarcinoma of the appendix, the malignant mucocele has little tendency to become locally invasive or spread widely, though such cases have been reported.[8] Pseudomyxoma peritonei, the development of mucinous material in the free peritoneal cavity, may follow rupture of a mucocele. While it is generally agreed that the malignant mucocele can produce this condition, the ruptured benign mucocele probably has the same potential. Pseudomyxoma peritonei can be produced in experimental animals in the absence of tumor.[43]

In most instances, careful local excision of the mucocele is adequate treatment. If local perforation has occurred with the development of a mucin-filled cyst, cure will generally be achieved by appendectomy plus removal of the adjacent cyst and its contents. Apparent cures of widespread pseudomyxoma peritonei are recorded with appendectomy and removal of the mucinous material.[27] Should the condition not be controlled with excision, massive ascites and adhesive bowel obstruction usually result.

CARCINOID TUMORS OF THE APPENDIX

Although carcinoid tumors of the appendix are relatively rare, most general surgeons will be confronted with at least one case during their surgical career. The incidence has been reported to be 0.32 per cent by the Mayo Clinic in a 30-year review of all appendectomy specimens at that institution.[39] The lesion represents the most common tumor of the appendix, composing at least half of all the tumors of that organ. It is usually reported to occur more commonly in females; however, this is probably a sampling error representing the greater numbers of female appendices examined in most pathology labs. This is due to the high rate of incidental appendectomy in females occuring during operations, such as cholecystectomy and hysterectomy. When a careful analysis is carried out, the rate of carcinoid is equal in both sexes.

The peak incidence is in the 3rd and 4th decade when argentaffin cell production is at its maximum. The low occurrence in the elderly may represent an actual degeneration of many of these tumors with age and thus explain the low autopsy incidence.

The tumor is probably most often found on routine pathologic examination of incidental appendectomy specimens, often not even being noted during the cursory gross inspection in the operating room. This fact is quite evident when one notes the marked increase in the incidence of the tumor when all appendices are examined histologically and not just grossly.[1] The remaining cases of appendiceal carcinoid present with a picture of either acute appendicitis or chronic appendicitis. The carcinoid will usually be found in the tip of the appendix and thus not really be involved in the pathogenesis of the inflammation. There are, however, cases where the carcinoid is directly involved in the luminal obstruction and thus is the underlying etiology of the appendicitis. Occurrence of the carcinoid syndrome itself is extremely rare, and usually requires the presence of liver metastases.

The main concern of the surgeon is the proper management of the problem if it is recognized in the operating room, or more commonly after the pathologist reviews the histologic slides. Almost all appendiceal carcinoids can be managed with appendectomy alone. Invasion of the serosa can often be identified; however, this does not change the prognosis. More radical surgery such as right hemicolectomy should be reserved for those few cases where there is evidence of lymph node metastases, local invasion of the cecum, or the uncommon case of a carcinoid greater than 2 cm in diameter. In a review of 1000 cases of appendiceal carcinoids managed with simple appendectomy, there have been only two cases with metastases.[39] Certainly more radical treatment of this disease is not indicated since the mortality rate from the operation would outweigh the small potential salvage.

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Blunt Torso Trauma

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As with most other areas of surgery, avoidable complications arising in the case of the injured may be broadly categorized as follows: (1) complications resulting from delay in diagnosis (preoperative), (2) complications arising from inadequate or inappropriate treatment, and (3) complications arising in spite of appropriate treatment. A comprehensive review of the myriad options avialable in the management of the injured patient will not be attempted. Rather, emphasis will be placed on those organs, injuries, and treatments which pose the greatest chance for improved results in the care of the injured patient.

COMPLICATIONS RESULTING FROM DELAY IN DIAGNOSIS

A thorough, comprehensive, and orderly evaluation of the injured patient is clearly the cornerstone on which all therapy must be based. Most patients who sustain blunt trauma to the torso are occupants of an auto, and the frequency of associated injuries to the head, neck, and extremities is noteworthy. Associated injuries, particularly those of the head and neck, often limit the value of certain diagnostic studies and alter the sequence and priority of therapeutic endeavors.

In patients with multiple injuries, the torso is most likely to harbor both the remedial injuries and those which pose the greatest diagnostic challenge. Since most blunt forces jeopardize simultaneously the organs of both the thoracic and abdominal cavities, the importance of thinking in terms of the "torso" in contrast to "thorax" or "abdomen" avoids the serious error of excessive preoccupation with what may prove to be the least important cavity. The steering wheel indenting the torso of the auto driver is a particularly good example of a mechanism creating simultaneous difficulties in both cavities with subtle and life-endangering implications[30] (Fig. 1). Diagnostic or therapeutic preoccupation with the hemoperitoneum arising from the fractured liver or ruptured spleen without simultaneous concern for the possibility of cardiac contusion or torn aorta is a common and serious error in diagnosis.

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Figure 1. Injuries to the automobile driver often include the head and neck (1), and lower extremities (3), in addition to the torso (2). The steering column or dashboard may simultaneously injure intrathoracic as well as intraabdominal viscera. (Reproduced from Freeark, R. J.: Abdominal trauma. *In* Hardy, J. O. (ed.): Rhoads' Textbook of Surgery, 5th ed. Philadelphia, J. B. Lippincott Co., 1977. Used with permission.)

Fortunately, most lung and chest wall injuries can be recognized by the careful interpretation of a good quality chest x-ray. Hemothorax, pneumothorax, rib fractures, and lung contusions are usually apparent and appropriate treatment can be instituted. The mediastinum and its contents are notoriously hard to evaluate by physical examination and portable chest radiography and are often the key to the management of life-endangering thoracic injuries. The history of rapid deceleration or of a direct blow to the anterior chest is the best and often the only clue to the presence of mediastinal injury. If, in addition, the patient exhibits unexplained shock, assymetry of blood pressure or pulse in the upper extremities, or fractures of the upper ribs, clavicle, or sternum, the index of suspicion of mediastinal injury is greatly increased. Additionally, if either the emergency portable AP x-ray view of the chest or the much more interpretable standard 6 meter PA view shows superior mediastinal widening, tracheal or esophageal displacement, pleural depression, or hemothorax, injury to mediastinal structures is likely. Angiographic study of the aortic arch is advisable and may quickly realign diagnostic or treatment priorities in such patients.

While most patients who sustain a rupture of the thoracic aorta will die immediately, an estimated 20 per cent of such patients will survive as the adventitia and fibrous sheath which surrounds the aorta maintain some continuity of the severed ends and limit the loss of blood with resultant hematoma formation.[22] Rupture of the hematoma into the pleural space occurs in an estimated 70 per cent of patients within the first 24 hours postinjury.[22] Once the hematoma ruptures, rapid exsanguination results and survival is rare. For these reasons, early recognition and treatment of the aortic rupture enjoy the highest priority in the care of the accident victim.

Since significant cardiac contusion occurs in close to 25 per cent of

patients with torn thoracic aorta and in an indeterminate number of anterior chest wall injuries, baseline EKG and enzyme studies should also be obtained and serially evaluated.[17, 29]

A third structure in the mediastinum that is occasionally injured in blunt trauma is the esophagus.[10] Here again, the initial chest x-ray may fail to demonstrate the existance of such an injury. Subsequent x-rays may reveal mediastinal air and confirmation of an esophageal injury will require Lipiodol swallow, endoscopy, or operative exploration.

If the mediastinum is the "silent" area of the thorax in which diagnosis is difficult and standard diagnostic measures insufficient, there are three such areas in the abdominal cavity.[7]

The first of these is the *intrathoracic portion of the abdominal cavity.* With both the liver and the spleen occupying locations behind the unyielding bony cage, it is not surprising that physical examination seldom confirms these injuries, and that sizable amounts of blood may be lost without visible abdominal distention.

The use of peritoneal lavage has largely resolved the diagnostic errors and delays in association with ruptures of the liver and spleen. Few significant injuries to either of these organs will be missed if the technique is carried out and interpreted as described by Root, Perry, et al.[5, 23] This is not the case with injuries to the diaphragm. Rupture of the diaphragm occurs most commonly on the left side, and when it occurs without other injuries, both peritoneal lavage and standard chest and abdominal x-rays are often negative.[9] On occasion, the only clue is elevation of the left hemidiaphragm or slight blunting or obliteration of the costophrenic angle on the involved side. Later as the intraabdominal organs are drawn into the chest, the patient may develop symptoms of gastric or intestinal obstruction. Although infrequent, rupture of the diaphragm is one of the most common injuries to escape diagnosis and result in serious late sequelae.

The second "silent area" in the abdominal cavity is the pelvis, in which the terminal small bowel, rectum, bladder, and urethra are organs vulnerable to injury. Here, errors in diagnosis are largely the result of failure to do a rectal examination, failure to pursue a complete diagnostic study in patients with microscopic hematuria, and failure to appreciate the sometimes subtle nature of injuries to the small intestine.

Urethral and bladder rupture commonly accompany pelvic fracture and are usually associated with gross hematuria or bloody drainage from the urethral meatus. It is less well appreciated that rupture of the distended bladder occurs as an isolated injury commonly following a "night on the town" in which the intoxicated driver elects to drive home with a full bladder. Such injuries may be associated with minimal peritoneal signs and only microscopic hematuria. They are easily missed if the only "cystogram" that is performed is that which accompanies intravenous pyelogram, particularly if a Foley catheter is in place and prevents marked distention of the opacified bladder. All patients with hematuria following trauma require evaluation of both the upper and lower urinary tract, and cystography should be performed with the in-

jection of at least 400 ml of contrast agent into the bladder followed by x-rays in both the AP and oblique projections, including a postevacuation series.

Tears in the mesentery or wall of the small intestine commonly accompany pelvic trauma. Rarely is free air noted on radiologic study, and in instances in which peritoneal lavage is employed, the lavage return is often only faintly blood tinged. Clear or pink lavage returns should be examined for bacteria and elevations of the WBC count if walled off intestinal perforations are not to be missed. It is also important to recognize that such injuries may be associated with the later development of symptoms of mechanical small bowel obstruction, as the mesenteric adhesions or contracting inflammatory process begin to compromise the small bowel lumen.

The third "silent area" of the abdomen is the retroperitoneal space. In addition to the kidneys and ureters, the importance of the duodenum and pancreas deserves particular emphasis. Injuries to these organs by blunt trauma are notoriously difficult to recognize, and particularly in the case of rupture of the 2nd and 3rd portions of the duodenum, carry the highest morbidity and mortality rates. Recognition of the lack of or delay in appearance of physical findings in most patients with these injuries is the first step in achieving a diagnosis. Serum amylase elevations are sometimes observed in such injuries and should be routinely and repeatedly checked. A plain radiograph of the abdomen of good quality may reveal the loss of one psoas shadow, duodenal ileus, lumbar spine fractures or scoliosis due to paravertebral muscle spasm, all of which suggest that the area has been subjected to a violent force. Such findings reinforce the need for assessment of duodenal integrity and should be followed by prompt contrast studies of the duodenum. Radiopaque contrast material is instilled into the stomach via nasogastric tube, and fluoroscopic evaluation of its course through the duodenum is carried out. In many instances, plain radiographs and peritoneal lavage will not detect the presence of retroperitoneal organ injury until late in the course. We favor surgical exploration or at the very least a contrast study of the duodenum, when the slightest risk of pancreatic or duodenal injury exists.

The location of silent areas and the more subtle organ injuries that occur within each of these torso regions is summarized in Table 1. It is hoped that recognition of these injuries by the special tests indicated will do much to eliminate the complications related to errors in diagnosis.

COMPLICATIONS RESULTING FROM INCORRECT TREATMENT

Complications arising as a result of inadequate or inappropriate treatment are becoming less frequent as the principles of management of organ injury become better established and more widely appreciated. Practices that once were commonplace have been shown to be unneces-

Table 1. *Clinically Silent Areas in Torso Trauma*

REGION	ORGANS INVOLVED	INJURY	SPECIAL TESTS
Mediastinum	Thoracic aorta	Tears	Arch angiogram
	Heart	Contusion	EKG, enzymes
	Esophagus	Rupture	Lipiodol swallow
Intrathoracic abdomen	Liver	Ruptures	Peritoneal lavage
	Spleen	Ruptures	Peritoneal lavage
	Diaphragm	Ruptures	Chest x-ray (repeated)
Pelvis	Bladder	Rupture	Cystography
	Small Intestine	Tear	Lavage (WBC's)
	Rectum	Tears	Digital rectal, proctoscopy
Retroperitoneum	Kidneys, Ureters	Fracture	IVP
	Pancreas	Transection, contusion	Amylase
	Duodenum	Rupture, intramural hematoma	Amylase, duodenography

sary or even harmful by controlled studies or the consensus of those with great experience.

Hepatic Injuries

The specific management of certain liver injuries remains controversial, yet general agreement can be found in regard to the operative treatment of the more common wounds. Direct suture ligation of bleeding parenchymal vessels is the preferred method of hemostasis in liver injuries which will not stop bleeding as a result of a period of gentle compression with temporary packs.[15] Hepatic artery ligation is a useful alternative in patients with lacerations which are inaccessible or involve the central region of one or both lobes.[1, 19] Bleeding is no longer controlled by the use of gauze packs or mass ligatures, and massive hepatic resections with their high mortality rate are currently employed largely as a last resort when other measures have failed to control bleeding. T-tube drainage of the common duct is no longer considered to minimize bile leakage but instead increases the risks of spesis, stress ulcer bleeding, and common duct injury.[16] Avoidable sequelae of liver injuries most often relate to inadequate drainage or other operative omissions.

1. *Inadequate perihepatic drainage* is the most commonly observed error in the management of liver trauma. As a consequence, perihepatic sepsis, subphrenic abscess, and fever of undetermined origin are common following these injuries. The inadequacies are usually a result of failure to place the drain site in a dependent location, failure to make an adequate opening in the abdominal parietes, and premature removal of the drains.

In the preoperative preparation of all patients with torso trauma, the posterior aspect of the lower chest and upper abdomen should be included in the skin preparation, and the patient should then be rolled back on to sterile drapes when returned to the supine position. This sterile field will encourage the use of a generous (3 finger) opening at the tip of or just caudal to the 12th rib as the ideal site to exit a number of large (1″) soft rubber drains which are placed in such a manner as to completely surround the injured liver for a period of 5 to 10 days. Gravitational forces and the reduced risk of evisceration through the drain site are strong reasons for emphasis on this posterior location. Every effort should be maintained to reduce the possibility that bacteria will enter the abdominal cavity as a result of contamination or unsterile technique associated with external drainage. The use of a commercially available draining wound management system is a notable advance in minimizing the inherent risks of contamination in prolonged abdominal drainage.

2. *Bile thorax* or the entrance of bile into the pleural space is usually a consequence of failure to repair associated diaphragmatic tears. Less frequent causes are neglected or improperly drained subphrenic infections and incorrect placement of thoracostomy tubes. The importance of identifying and repairing all diaphragmatic defects at the time of laparotomy is emphasized. Thoracentesis or tube thoracostomy should seldom be performed below the level of the 5th interspace, thereby avoiding inadvertent subdiaphragmatic penetration.

3. *Hemobilia* or hemorrhage into the biliary tree usually results from failure to identify or adequately treat a central cavity within the liver. Such pockets of bile, blood clot, and necrotic liver under pressure may lead to erosion of vascular structures and creation of a false aneurysm early or many months after the injury with the shed blood finding its way into the biliary radicals. A clinical triad of upper gastrointestinal bleeding, biliary colic, and obstructive jaundice then results.[26] The capsule should rarely be closed in liver injuries, and meticulous hemostasis and adequate drainage at the time of initial repair will go a long way to prevent the development of this serious complication of hepatic trauma. Insertion of T-tubes into the common duct to identify areas of ductal disruption, decompress the biliary system, or monitor for the presence of this rare complication is seldom justified. Small central hematomas may be observed and followed by serial liver scans but large cavities should be entered, bleeding controlled, and external drainage provided.

Hepatic abscess is relatively infrequent following hepatic trauma and should raise the question of a retained foreign body or inadequate provision for egress of bile, blood, or necrotic tissue from the deeper areas of injury.

Pancreatic Complications

Pseudocyst and abscess formation, delayed hemorrhage, pancreatitis, and pancreatic fistula are far more likely to follow pancreatic injuries in which some form of sump drainage was not employed.[3] The failure of soft rubber drains to prevent peripancreatic collections in some

patients, and the need to provide large external drain sties in a location where the firmer sump drains will not erode adjacent bowel or major blood vessels has led to greater use of the bed of the 12th rib posteriorly for the site of exit of most subdiaphragmatic drains.

The need for careful inspection of the injured pancreas at the time of laparotomy deserves emphasis. This can ordinarily be accomplished by thorough mobilization of the lateral and gastroduodenal attachments of the hepatic flexure of the colon, thereby exposing the head and neck area and permitting entrance into the lesser omental bursa by way of the "front door." Upward retraction of the stomach and downward displacement of the transverse mesocolon, particularly after the lateral portions of the greater omentum are detached from the transverse colon, provides unparalleled opportunities to inspect the pancreas, duodenum, posterior stomach, and splenic hilum. Minimal dissection along the 2nd and 3rd portions of the duodenum will ordinarily provide access to the uncinate process and superior mesenteric vessels. If disruption of the main pancreatic duct is confirmed or appears likely in association with pancreatic injuries, the choice of operative treatment rests with resection of the pancreas distal to the ductal disruption, drainage of the distal portion into a Roux-en-Y loop of jejunum, or merely sump suction drainage. The latter approach carries a greater likelihood or requiring a secondary operation at a later date, whereas distal pancreatectomy may be followed by the development of exocrine or endocrine pancreatic insufficiency.[8]

When sump drainage of the pancreas is employed for treatment of ductal disruption, diffuse post-traumatic pancreatitis, pancreatic abscess, or phlegmon, it is imperative that the firm rubber or plastic catheter lay in close proximity to the area of injury and exit from the body in such a manner that pressure erosion of small bowel, colon, or major vessels is avoided. Such a catheter should remain in place (frequently surrounded by additional soft rubber drains) for a minimum of 5 days.

Cannulation and/or suture repair of the severed pancreatic duct, pancreaticoduodenectomy, and onlay pancreaticojejunostomy are procedures employed by experienced surgeons under special circumstances, and their use is properly limited.

Duodenal Injuries

Duodenal hematomas are being recognized preoperatively with increasing frequency, and when they appear to exist as an isolated injury are often managed nonoperatively. When encountered at surgery, they should not be misinterpreted as hemorrhagic infarction of the duodenum but are readily evacuated through a seromuscular incision with gentle expulsion of the contained clots and liquid blood. Gastroenterostomy is seldom necessary as an adjunctive procedure, thereby avoiding the not infrequent complications of stomal dysfunction or the risk of late anastomotic ulcers.

Duodenal rutpure usually responds to a meticulous suture repair, unless the diagnosis is delayed over 8 hours or major pancreatic injury

accompanies it.[17] Operations like the Whipple procedure should rarely be necessary, but recourse to serosal patch techniques and to gastrotomy with decompressive and feeding jejunostomies is frequently advisable when repair appears precarious. Each of these procedures carries with it a number of potential sequelae that serve to emphasize the importance of early diagnosis and repair at a time when recourse to special techniques will not prove necessary.

Spleen

Operative removal of the injured spleen is advisable in adult patients with gross anatomic disruptions of the splenic pulp or persistent bleeding from a capsular tear. Efforts to preserve immunologic functions of the spleen appear advisable in small children in whom bleeding can be controlled and careful observation is possible.[4]

Avoidance of complications arising from removal of the spleen requires an appreciation of the close proximity of the greater curvature of the stomach (and its short gastric vessels), the tail of the pancreas, the splenic flexure of the colon, and the left adrenal gland. Mobilization of the spleen along with the body and tail of the pancreas from the underlying renal (Gerota's) fascia is the preferred technique of splenectomy in patients with traumatic injuries. The avascular plane is entered followwing incision of the spleenorenal ligament, and the left upper quadrant viscera anterior to the kidney are delivered into the operative wound to allow for meticulous control of the hilar and short gastric vessels.

Drainage of the splenic bed is employed following splenectomy only if hemostasis proves inadequate, or there is associated injury to the pancreas. Under these circumstances, drains exit posteriorly at the level of the 12th rib. Routine drainage of the splenic bed appears to increase the likelihood of subphrenic infection. Even in the face of fecal contamination of the splenic bed, the benefit of drainage is uncertain, and it appears likely that meticulous hemostasis and thorough lavage is more effective in reducing the incidence of subphrenic infection.

Retroperitoneal Hematoma

The management of retroperitoneal hematomas should be carefully individualized on the basis of their location, expansile quality, and findings on preoperative I.V.P. or angiography. Hematomas in the vicinity of the duodenum, head and body of the pancreas, or base of the transverse mesocolon should be entered and carefully explored for evidence of underlying pancreatic or duodenal injury. Operative exploration is also advisable for hematomas which are large in size and rapidly expanding, or where an underlying arterial injury is suspected on the basis of a pulsatile quality, bruit, thrill, or prior angiogram. In penetrating wounds, the integrity of important structures such as the ureter in the vicinity of the hematoma must be ascertained.

However, most paravertebral, perirenal, intramesenteric, and pelvic hematomas that arise in conjunction with blunt trauma arise from venous or arterial sources that stop spontaneously or by the tamponade

which the overlying peritoneum or hematoma provide. To explore these hematomas invites a risk of continued bleeding which, particularly in the case of the hematoma associated with pelvic fracture, may be difficult if not impossible to control.

Hematomas behind the ascending or descending colon are usually related to renal injury and can be safely ignored if the status of the kidneys has been evaluated preoperatively. Exploration or evacuation, particularly when it involves opening Gerota's fasica, is often followed by brisk hemorrhage that has led to needless sacrifice of the involved kidney. Since most renal injuries heal uneventfully if left alone, it is our practice not to open Gerota's fascia unless the preoperative studies (I.V.P., renal angiography) confirm a major hilar disruption or vascular injury, and additionally, the renal artery and vein are first controlled by linen tourniquet tapes.

Pelvic hematomas accompanying pelvic fractures are among the most difficult to evaluate and treat. Adequate volume replacement and careful study of the lower urinary tract and rectum must precede operative intervention. Nonoperative treatment is much preferred when there is no evidence of other injuries requiring operative intervention. Once surgery is elected, if at all possible, the pelvic peritoneum should be kept intact for its tamponading effect upon bleeding from the pelvic venous plexus. Assuring the integrity of the distal small bowel is an operative responsibility, but the integrity of the rectum and bladder should be evaluated *prior* to surgery. Bladder repairs and suprapubic cystostomy can usually be achieved without release of the pelvic hematoma. If rupture of the hematoma does occur and is followed by persistent and exsanguinating blood loss, consideration can be given to attempts at suture ligation of the pelvic veins, or in desperation, a gauze pack may be required. While an arterial source of blood loss is infrequent in these injuries, successful embolization or ligation of the responsible arteries has controlled bleeding often enough to justify angiographic study and angiographic or operative interruption when arterial sources are confirmed.[25] Nonoperative measures including vigorous blood replacement until spontaneous control occurs are frequently better alternatives.

Renal Injuries

Avoidable complications of renal inuuries are usually a consequence of failure to adequately study the injured urinary tract prior to surgery or of unwise attempts to inspect or repair the injured kidney at the time of laparotomy. Knowledge of the magnitude of renal injury as well as the presence of a functioning kidney on the opposite side is an esential prerequisite to the management of renal trauma and is gained largely by preoperative infusion pyelography occasionally supplemented by renal angiography. Since the overwhelming majority of renal injuries stop bleeding spontaneously and heal without serious sequelae, it is rarely advisable to open Gerota's fascia at the time of laparotomy, particularly without prior dissection and control of the renal vessels. Failure to heed this advice often results in brisk bleeding from the injured kidney which is then sacrificed in a desperate attempt to limit further blood loss.

Under circumstances where preoperative studies have confirmed a vascular injury to renal pedicle or gross disruption of renal structures with extrarenal extravasation of contrast material making operative inspection and repair advisable, liberal posterior flank drainage in the tip of the 12th rib is essential.

Lower Urinary Tract Injuries

Complications arising from the repair of the injured ureter or bladder can usually be avoided by precise surgical repair and adequate external drainage. Most wounds to these structures can be repaired using fine absorbable suture such as chromic catgut or polyglycolic sutures. Proximal decompression of major ureteral repairs is usually advisable and may be achieved by nephrostomy, proximal ureterostomy with flank drainage, or the insertion of a ureteral catheter that is passed proximally and distally into the bladder for later cystoscopic retrieval.

Major bladder injuries should be closed in layers with care to avoid compromise of the lumen of the terminal ureters. Insertion of a subrapubic cystoscopy tube assures continuous decompression of the bladder during healing, and ready evacuation of clots without risking the greater likelihood of sepsis or epididymitis from indwelling urethral catheter drainage.

Injuries to the male urethra require urologic consultation (and preoperative urethrography) particularly where a Foley catheter cannot be easily inserted into the bladder. While the presence of blood in the urethral meatus should always suggest the possiblity of urethral disruption, gentle passage of a sterile urethral catheter is justified prior to urethrography provided the effort is abandoned should any degree of obstruction be encountered.[2] Once the catheter enters the urinary bladder, most partial urethral tears will heal with the catheter acting as a splint to maintain the urethral lumen.

Gastrointestinal Injuries

Blunt or penetrating injuries to the abdominal esophagus, stomach, duodenum, or small intestine seldom pose serious risk of complications when promptly discovered and properly repaired. Late or missed injuries are prone to leakage, fistula formation, advanced peritonitis, retroperitonitis, intraabdominal abscess, ileus, mechanical obstruction, and evisceration. Avoidable pitfalls are overlooking perforations in the mesenteric portions of the viscus, excessive turning in of cut edges of bowel wall with lumenal compromise and obstruction, and ill advised attempts to resect and anastomose injured bowel rather than undertaking simple repair. Intraperitoneal drainage and exteriorization are rarely if ever advisable for the injured stomach or jejunoileum. Prolonged nasogastric or long tube decompression may be advisable. In patients with advanced peritonitis and mechanical obstruction following such injuries, meticulous surgical debridement, long tube intubation, and copious lavage of the peritoneal cavity, as recently advocated by Hudspeth, appear worthy of consideration.[12]

Colon Injuries

The injured intraperitoneal colon continues to provoke the greatest controversy in regard to operative treatment with a broad range of therapeutic possibilities endorsed by some and considered hazardous and fraught with complicatons by others.[18, 28]

Primary repair appears safe in patients with favorable wounds (stab and low-velocity gunshot wounds) that are operated within 2 hours after injury and receive vigorous preoperative antibiotic therapy.[18] Under these circumstances both the right and left halves of the colon are treated in a manner similar to the small intestine. Even under these highly favorable conditions, repair and "drop back" appears unwise if there is much fecal spill or other serious intraabdominal injuries such as those involving the duodenum, pancreas, liver, or kidneys.

Colon wounds arising under other than highly favorable conditions (treatment delayed over 4 hours, other serious intraabdominal injury, high-velocity gunshot, blunt trauma, gross fecal contamination, major mesenteric disruption), whether involving either the right or left halves of the colon are best managed by exteriorization over a plastic rod or repair with a proximal colostomy. So-called early return of the repaired and exteriorized colon may shorten hospital stay but should not affect the key decision for or against exteriorization.[21]

Rectal Injuries

Injuries to the rectum are infrequent in blunt trauma to the torso but occasionally accompany violent pelvic fractures and disarticulation injuries to the perineum and hip regions. Once confirmed, only the most vigorous treatment may be lifesaving. Half-hearted efforts at drainge, attempts to spare the patient a temporary colostomy, and undue reliance on antibiotics are common errors in the mangement of these wounds. Suture repair, completely diverting proximal colostomy, perirectal or presacral drainage, and transanal evacuation of the contents of the defunctionalized segment are the only safe methods to manage the majority of these injuries.[14]

The technique and complications of colostomy are dealt with elsewhere in these issues. It is the author's prejudice that loop colostomy is adequate for most colon injuries, but that rectal wounds require a divided "double barrelled" colsotomy. In both situations, a site other than the primary incision and as near as possible proximal to the repaired segment is preferred.

UNAVOIDABLE COMPLICATIONS

When one considers the variety and magnitude of abdominal injuries and the circumstances which arise to delay or modify ideal treatment, it is not surprising that a certain number of complications occur in spite of ideal surgical management. While none of these conditions

can properly be considered "inevitable" nor are they in any sense acceptable, we must concede that at the present state of our knowledge, many are unavoidable.

Peritonitis, Intraabdominal Abscess

Traumatic perforation of the gastrointestinal canal leads to leakage of organisms into the peritoneal cavity and retroperitoneal space with variable degrees of peritonitis. Studies of human and animal peritoneum have confirmed its remarkable ability to successfully clear large numbers of bacteria by three mechanisms: (1) the direct absorption of particles, including bacteria from its extensive peritoneal surface area, (2) phagocytosis of bacteria by polymorphonuclear leucocytes, (3) confinement of infection by the formation of fibrinous adhesions. Abscess formation and lethal consequences of peritonitis are far more likely to occur in the presence of foreign material, necrotic tissue, and adjuvant substances such as hemoglobin, bile salts, and gastric mucin.[11]

The beneficial effects of antibiotics on the course of peritonitis is well established. The excellent blood supply of the peritoneum assures a high concentration of systemically administered antibiotics on peritoneal surfaces, but many favor its local instillation into the peritoneal cavity additionally. To be effective the systemic antibiotic should be administered as close to the time of injury as possible, and benefits are hard to confirm if more than several hours have elapsed before treatment is instituted. When perforative peritonitis seems likely (e.g., in all gunshot wounds of the abdomen and stab wounds or blunt trauma with obvious peritonitis), we begin broad-spectrum intravenous antibiotic therapy with agents effective against aerobes as well as anaerobes as soon after as the patient reaches the emergency department as possible. The broad aerobic and anaerobic spectrum of chloromycetin and its low incidence of side effects, particularly when administered parenterally make it a regular part of our current antibiotic therapy. Operative control of the site of perforation is accomplished once detected. This is initially achieved with atraumatic intestinal clamps until complete exploration has established the proper priority of treatment for all injuries identified. It is surprising how often the inexperienced trauma surgeon will begin suturing "holes" before he has completed a thorough investigation and evaluation of other possible injuries. Initially and at the completion of the operative procedure, the entire peritoneal cavity is thoroughly irrigated with copious amounts of temperature-controlled balanced salt solution to remove foreign material, blood, and fibrinous exudate. While the benefits of antibotic instillation as well as continuous peritoneal lavage are still under debate, it appears inadvisable to allow significant amounts of any solution to remain within the peritoneal cavity or in its recesses or gutters. Therefore, emphasis is placed on thorough aspiration of the irrigating solution, rather than allowing a residue of solution with or without an antibiotic which might impair the absorptive or phagocytic activity of the peritoneal lining.

In spite of these efforts, severe sepsis and intraabdominal abscess formation continue to occur following abdominal trauma. The former is

a major factor in the development of the adult respiratory distress syndrome, stress ulceration of the gastrointestinal tract, post-traumatic hepatic dysfunction, both oliguric and nonoliguric renal failure, impaired cardiac function, and a spectrum of coagulation disorders including disseminated intravascular clotting.

The overall treatment of these specific complications of sepsis will not be reviewed. The need for early identification and drainage of intraabdominal abscess is, however, of great importance. A vigorous diagnostic approach to patients with fever following abdominal trauma pays many dividends in assuring a prompt and uncomplicated convalescence. In addition to a careful examination of the chest, abdomen, and pelvic areas, the WBC, differential hemoglobin concentration, and platelets should be serially assessed. Early and repeated use of gallium and technetium scans, supplemented by ultrasonic study when feasible, have done much to assist in the confirmation and drainage of these localized abscesses within the abdominal cavity. Since gallium concentrates at sites of protein leak and polymorphonuclear leucocytic concentration, a "hot spot" may represent the abdominal incision, a site of recent surgery, or an area of cellulitis, phlegmon, or abscess formation. Confirmation of the presence of a sonolucent area consistent with abscess formation by means of ultrasound is helpful but may not be possible if open wounds, dressings, colostomy stomas, or gaseous distention preclude a diagnostic study. When the clinical condition of the patient suggests the possiblity of an intraabdominal abscess, it is usually wiser to reexplore the abdomen rather than to await a "diagnostic" study or rely upon a change of antibiotics or further delays to resolve the dilemma. Once exploration is decided upon in most instances, a formal laparotomy is required to confirm unrecognized etiologic factors, to assure identification of all abscess cavities, and to provide adequate and complete drainage. Rarely is the extraserous approach to intraabdominal sepsis a wise one for patients in whom the inflammatory process followed abdominal injury.

Postoperative Fever and Wound Infection

The great length of the incision employed in the usual laparotomy for abdominal trauma and its regular and heavy contamination by bacteria arising from the intraperitoneal injuries make a certain number of incisional infections inevitable. Strict adherence to a policy of never closing the skin and subcutaneous layers of the contaminated abdominal wound will go a long way toward lowering the incidence of this complication. Those with a regrettable compulsion to close the skin in such wounds have found irrigation with antibiotics or iodine solution, and suction catheter drainage, to be advantageous in reducing the incidence. Our own preference is for gauze packing applied loosely to the level of the fascia with daily changes until the fifth to seventh postoperative day, at which time the clean granulating surfaces can usually be approximated using sterile microporous paper tapes. With delayed primary closure, the final scar is usually quite acceptable cosmetically, and the important fascial layer is allowed to heal without an overlying pool of

pus bathing the suture line. Additionally, the wound is excluded as one potential source of postoperative fever.

Since investigation of the source of fever following surgery of abdominal trauma occupies a considerable portion of the surgeon's time as well as the hospital diagnostic facilities, it is perhaps worthwhile to emphasize the importance of excluding as many extraabdominal sources as possible. Early removal of or frequent rotation of indwelling plastic venous or arterial catheters is necessary to reduce the risks of thromboembolism or catheter sepsis. The Foley catheter should always be inserted with gentle aseptic technique and should be removed as soon as feasible. The connecting drainage line and receptable should remain a closed system for as long as the catheter remains in place. The high risk of atelectasis and pneumonia, especially following surgery or infection underneath the diaphragm, places a great premium on vigorous respiratory toilet and physical therapy early in the postoperative period, so that this source of fever, pleural effusion and diaphragmatic distortion can be more readily distinguished from the development of a subphrenic abscess. Careful attention to medications is also advisable, as more than an occasional fever of undetermined origin has been traced to an allergic or idiosyncratic reaction to one of the antibiotics or other medicines the patient is receiving.

Fistula Formation

Persistent drainage of bile, pancreatic or gastrointestinal contents may follow injuries to the respective organs in spite of appropriate surgical efforts. Where drains were employed, as in hepatic and pancreatic injuries, they should be slightly withdrawn or replaced by a firm catheter that will assure free flow out the fistulous tract while at the same time the drain itself is not in direct contact with the fistula's site of origin.

In fistulas involving the gastrointestinal tract, the potential for major fluid and electrolyte losses, activated digestive enzymes, and the greater likelihood of the external opening appearing within the abdominal incision makes management more difficult.

Several principles should guide the management of such fistulae.

1. The site, size, and origin of the fistulous tract should be confirmed by injection of contrast material into the external opening with appropriate flouroscopy and two-dimensional radiographs.
2. Collection, measurement, and occasionally biochemical analysis of the fistulous drainage should be carried out over a 24-hour period to confirm its origin and assess its implications for replacement therapy. Continuous suction applied to a multiholed catheter near or just inside the external opening will facilitate such studies, protect the surrounding skin and wounds, and minimize the need for dressing changes.
3. Fistulas arising from the gastrointestinal, urinary, or pancreaticobiliary structures tend to close spontaneously as long as distal obstruction is not present and foreign bodies and abnormal tissue are not present in the fistulous tract. In the trauma patient, the possibility of a retained foreign body (missile, drain, gauze sponge) should always be considered.
4. Fistulous output will ordinarily diminish with restriction of all oral intake or alternatively the use of an elemental diet.

5. Operative intervention to close a fistula is seldom required for liver or pancreatic injuries and is neither necessary or likely to succeed in early fistulas involving the gastrointestinal tract. The advent of parenteral nutritional support has generally provided a better solution to the nutritional and fluid and electrolyte needs of such patients. Fistulous outputs of greater than 3000 ml per 24 hours are difficult to manage for extended periods by nonoperative measures and may require operative treatment.
6. The presence of significant temperature elevations in a patient with a fistula should suggest the possibility of incomplete drainage due to obstruction in the fistulous tract or intraabdominal abscess formation near or remote from the fistula tract.

Postsplenectomy Complications

Left lower lobe atalectasis and pneumonia are the most common complications following splenectomy. Prevention with vigorous respiratory physical therapy and tracheobronchial toilet is particularly important, so that the accompanying elevations of the diaphragm and blunting of the costophrenic angle will not be confused with subphrenic sequelae. It is our practice to institute nasotracheal catheterization 12 hours following all procedures which involve the immediate subdiaphragmatic recesses in an effort to avoid this complication.

Thrombocytosis with platelet counts in excess of 300,000 per cu mm follows splenectomy in the majority of patients. Its relationship to a purported increased incidence of thromboembolic complications remains uncertain. It has been our practice to encourage leg elevation and early ambulation in all patients whenever feasible, but prophylactic anticoagulation has rarely been employed in the post trauma patient.

Steele and Lim have reported a four to six fold increase in the rate of infectious sequelae following splenectomy.[27] The majority of these infections were respiratory in origin, although a higher incidence of wound infection was also noted. An increased susceptibility to septicemia following splenectomy has been documented in children under 2 years of age.[6] A reported 1 per cent incidence of fatal sepsis after splenectomy for trauma in children of all ages had led some authorities to undertake splenic repairs, partial splenectomy, or long-term antibiotic prophylaxis when splenctomy was required.[20] At the present time, we adivse splenectomy for injuries that do not stop bleeding spontaneously or with the application of local hemostatic agents, and penicillin prophylaxis for splenectomized children up to the age of 15. An alternative approach is to adivse the child's parents of the need for immediate and vigorous medical attention at the first sign of infection.[24]

Posthepatic Injury

A variety of complications seem unavoidable following the surgery of hepatic trauma.

Pleural effusions usually sterile and bile free are not infrequent following major injuries or hepatic resections. Repeated aspirations by thoracentesis performed in the lateral decubitus position with the needle inserted at no lower than the 5th interspace will usually evacuate the pleural fluid and avoid the risk of needle entry into the subphrenic space.

Gastrointestinal bleeding, usually as a result of stress ulcers on the stomach, appears to be more common in patients with hepatic trauma. Other etiologic possibilities include esophageal varices, duodenal ulcer, or hemobilia and are usually best resolved by endoscopy. Prophylaxis of stress ulcer bleeding with antacid buffering of gastric juice is advisable and should be employed along with periodic gastric decompression. Surgical intervention to control relentless bleeding is usually delayed until the magnitude of blood loss approaches 3000 ml and all abnormalities of the coagulation profile are corrected. We favor truncal vagotomy, pyloroplasty, and suture ligation of bleeding sites in the usual stress ulcer patient.

SUMMARY AND CONCLUSIONS

Most blunt forces pose a simultaneous hazard to the regions of the chest and abdomen. Complications following such injuries to the torso are usually the result of delays in diagnosis or inadequate operative treatment. Clinically silent regions in the chest and abdomen where such injuries are difficult to detect make the need for special diagnostic studies essential. Successful operative treatment requires accurate hemostasis, detection of all injuries, and the application of generally accepted principles of repair. In spite of the best such efforts, certain complications will continue to be encountered, and their control and treatment remain a challenge to all who care for the injured.

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Hepatic Complications

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Pathophysiologic alterations in liver function during and after major surgical procedures occur in the following three circumstances: (1) alterations in the function of normal liver surrounding general surgical procedures, (2) exacerbation of pre-existing pathophysiology in patients with known liver disease by general surgical intervention, and (3) alterations in hepatic function following operative intervention directly on the liver itself.

Accordingly, the ensuing discussion will deal sequentially with each of these subheadings. It should, however, be emphasized that a significant overlap exists amongst this artificial separation of clinical categories of hepatic malfunction.

HEPATIC DYSFUNCTIONS FOLLOWING GENERAL SURGICAL PROCEDURES

Both general anesthesia and surgical intervention invariably cause a minor degree of hepatic insult to all patients.[24, 73] These alterations are usually self-limited and do not alter the patient's postoperative course. Transient elevations in transaminase levels occur in most patients. They are significantly higher in upper abdominal surgery than in other types of surgery. Operations involving halogenated anesthetics result in a more severe and often prolonged elevation in enzyme levels than when using other types of anesthetics.[71, 73] For the most part, elevations in serum bilirubin and alkaline phosphatase levels do not occur as a result of these minor insults. When transaminase levels are persistently elevated for more than a few days, especially if associated with elevated bilirubin and alkaline phosphatase values, then serious hepatic injury must be considered. Since jaundice is the most common and significant

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Table 1. *Classification of Postoperative Jaundice*

PREHEPATIC JAUNDICE	HEPATOCELLULAR JAUNDICE	POSTHEPATIC JAUNDICE
Hemolysis { Transfusion, Congenital, Acquired	Shock: hypotension, hypoxia	Bile duct ligation and stricture
Hematoma	Drug hepatitis	Choledocholithiasis
Bile leak	Viral hepatitis	Pancreatitis
Sepsis	Hepatic artery ligation	Postoperative cholecystitis
Benign postoperative cholestasis	Sepsis	
Pulmonary embolism	Benign postoperative cholestasis	
	Gilbert's disease	
	Dubin-Johnson syndrome	

clinical manifestation of serious hepatic dysfunction, this section will be best discussed from the viewpoint of postoperative jaundice.

POSTOPERATIVE JAUNDICE

Postoperative jaundice is a difficult diagnostic and therapeutic challenge even for the most astute hepatologist. It is often a consequence of multiple factors acting in consort. In each case, the following factors must be carefully evaluated and re-evaluated: (1) preoperative hepatic status, (2) history of alcohol abuse, toxic drug exposure, or possible contact with hepatitis, (3) type and duration of operative procedure, (4) type of anesthetic agent used and delineation of all other medications, (5) number of blood transfusions and other blood products, (6) transient low cardiac output or hypoxia during or after operation, (7) intraoperative findings, (8) pre-existing medical conditions such as hemolytic disease and congestive heart failure, and (9) possibility of occult sepsis.

Generally, one of the following three basic mechanisms is dominant and responsible for the jaundice. These are: (1) prehepatic component (pigment overload), (2) hepatic component (impaired hepatocellular function), and (3) posthepatic component (bile duct obstruction). Table 1 enumerates the common causes of jaundice of each type. A diagnosis of the mechanism most responsible is essential since the disease may be self-limited on one hand, may require surgical intervention for bile duct obstruction on the other, and alternately may be an indication of sepsis or fulminating hepatic failure.

Prehepatic Jaundice

Five possible sources of pigment overload occur in the postoperative period: (1) hemolysis of transfused blood (either old or mismatched), (2) spontaneous hemolysis of the patient's own blood due to intrinsic erythrocyte defects (sickle cell anemia, thalassemia, glucose-6-phosphate dehydrogenase deficiency, and others), (3) hemolysis due to an autoimmune Coombs-positive hemolytic anemia, (4) hemolysis due to sepsis (Clostridia, E. coli, Streptococcus), and (5) reabsorption of extravasated blood and/or bile. In the presence of hemolytic jaundice, the

majority of increased serum bilirubin is unconjugated while transaminases and alkaline phosphatase values are normal (unless concurrent hepatobiliary disease is present). Further, serum haptoglobin levels are markedly reduced or absent, and reticulocytosis and hemosiderinuria are present.

Ten per cent of transfused erythrocytes which are 10 days old will be destroyed within 24 hours of transfusion. One unit of such blood yields 250 mg of bilirubin, which is approximately the amount of bilirubin produced daily in the normal individual. If 10 units of blood are transfused during or after surgery, the bilirubin load is ten times normal (2.5 gm per day), which most certainly will produce clinical jaundice.

Hemolysis in patients with hemoglobinopathy, triggered by operative insult (hypoxia or pharmacologic agents), occasionally can raise the serum bilirubin level up to 20 mg per 100 ml or greater. Jaundice due to hemolysis and due to reabsorption of extravasated blood and bile is self-limited and is most often successfully treated medically. However, a high index of suspicion for combined disease must be considered in hemolytic jaundice since the incidence of pigment biliary stones is high in patients with hemoglobinopathies. Should a large fraction of high serum bilirubin be conjugated with marked evaluation of alkaline phosphatase, endoscopic retrograde cholangiography should be performed to rule out a posthepatic component. Extravasated blood and bile can become infected and may lead to sepsis. Systemic sepsis may not only result in hemolytic jaundice but also result in a serious hepatocellular injury, presenting with elevated values of transaminases. Aggressive treatment of systemic infection when it occurs, and early treatment of intra-abdominal abscesses and biliary leakage will avert the occurrence of serious sepsis and minimize the reabsorption of bile and old blood.

Hepatocellular Jaundice

The most common causes of postoperative hepatocellular jaundice are: (1) episodes of hypotension and/or hypoxia before, during, or after surgery, (2) use of hepatotoxic drugs including hepatotoxicity from general anesthetics, (3) sepsis, (4) viral hepatitis, (5) hepatic artery ligation (accidental or intentional), (6) obscure benign postoperative cholestasis, and (7) rare familial hyperbilirubinemia.

Hepatocellular jaundice typically presents with markedly elevated transaminase levels. However, drug-induced hepatocellular injury often results in prolonged jaundice associated with high alkaline phosphatase levels and only minimal elevations in hepatic transaminase values. This chemical picture of "cholestatic jaundice" is extremely difficult to differentiate from that of extrahepatic biliary duct obstruction. Visualization of the biliary duct by transhepatic needle cholangiography or endoscopic retrograde cholangiography are mandatory prior to re-exploration. A needle biopsy of the liver is helpful to distinguish hepatocellular jaundice, but is seldom diagnostic for extrahepatic biliary duct obstruction. Some of the most commonly used hepatotoxic drugs are listed in Table 2. Further, so-called "benign postoperative cholestasis"[66] is also associated

Table 2. *Drugs Reported to Cause Hepatic Injury*

Anesthetics	*Antituberculous*
Halothane	Isoniazid
Methoxyflurane	PAS
	Ethionamide
Antibiotics	
Erythromycin	*Hormones*
Tetracycline	Methyltestosterone
Sulfonamides	Synthetic estrogens
Penicillin	Synthetic progestational agents
Anti-inflammatory	*Psychotropic Drugs*
Acetaminophen	Phenothiazine
Indomethacin	MAO inhibitor
Phenylbutazone	Tricyclic antidepressants
Probenecid	
Salicylates	*Miscellaneous*
	Alpha-methyldopa
Antimetabolites	Diphenylhydantoin
Azathioprine	Oxypenisatin
Chlorambucil	Propylthiouracil
6-MP	Methimazole
Methotrexate	Sulfonureas
	Chlorpropamide
	Thiazide diuretics

with increased levels of alkaline phosphatase and only mild elevations in transaminases. Typically, this cholestatic jaundice follows a major operative procedure on a poor risk patient who has received multiple transfusions, it appears soon after surgery, and may last for a week or longer.

There are two types of familial hyperbilirubinemia which are exacerbated by surgical stress, infection, or fasting. They should be also considered as hepatocellular jaundice. Exacerbation of Gilbert's disease results in elevations of unconjugated serum bilirubin presenting the chemical picture of hemolytic jaundice. Dubin-Johnson syndrome produces conjugated hyperbilirubinemia and is characterized by the inability to excrete cholecystographic dye and bromsulphalein.

It is essential to recognize hepatocellular injury early postoperatively in order to provide adequate therapy as well as to avoid erroneous surgery. Supportive therapy must be initiated as soon as hepatocellular disease is recognized, and certainly, discontinuation of hepatotoxic drugs is mandatory should they be present. Management of aggressive hepatic insufficiency will be discussed in later sections.

Posthepatic Jaundice (Bile Duct Obstruction)

With rare exceptions, extrahepatic biliary duct obstruction is the only type of postoperative jaundice which requires reoperation. The obstruction may be complete and constant or it may be partial and intermittent. Retained or undiagnosed common duct stones, accidental ligation or stricture of the bile duct, and extrinsic bile duct obstruction due

to postoperative pancreatitis are the common causes. In posthepatic jaundice, the majority of increased serum bilirubin is conjugated and the alkaline phosphatase is elevated, while the values of serum transaminases are usually only modestly elevated. Early diagnosis of biliary duct obstruction is advisable in order to maintain the risk of reoperation at a minimum. If not recognized early, biliary duct obstruction may result in cholangitis, sepsis, and progressive deterioration of the patient due to combined nutritional and metabolic abnormalities, and eventually it may lead to biliary cirrhosis. These intercurrent events provide the increment in risk when operative intervention is being made. However, reoperation on the jaundiced patient is extremely risk-laden since the patient may already have a significant hepatocellular dysfunction and may not have recovered from previous operative insults to other vital organs. Unnecessary additional operative intervention must be avoided. Early endoscopic retrograde cholangiography or transhepatic needle cholangiography should be performed when the diagnosis of extrahepatic biliary obstruction is first considered. Heretofore, percutaneous transhepatic cholangiography has been practiced only when the patient can proceed to the laparotomy without delay, because of frequent complications.[25] A modification of this technique by Chiba group in Japan has decreased the complication rate to a minimum. The "skinny" Chiba needle technique was first introduced to American literature by Redeker et al.,[60] who reconfirmed the safety of the procedure. Endoscopic retrograde cholangiography also carries a minimum of risk of less than 0.5 per cent complication rate.[69] With these newer techniques the biliary duct system can be visualized in 90 per cent of all jaundiced patients and practically in all patients with biliary duct obstruction.[16, 60]

POSTOPERATIVE COMPLICATIONS IN PATIENTS WITH LIVER DISEASE

Certainly, all the disorders which may occur to the patient with a normal liver after general surgery might very well occur to a similar patient with known liver disease. Each of these complications should be expected to result in a more serious prognosis in a patient with diminished hepatic reserve. It is, therefore, essential for the physician to adequately determine the extent of liver disease prior to recommendation for surgery in this group of patients. When surgery is contemplated for patients who have abnormal liver function tests, it is best to delay elective surgery until the etiology of liver dysfunction is determined and liver function tests return to normal. Transient abnormalities in liver function tests are often associated with pancreatitis, attacks of cholecystitis and biliary colic, or inflammatory bowel diseases. In these conditions, however, necessary surgery should not be delayed merely because of abnormal liver function tests. If a diagnosis of acute viral hepatitis[27] is confirmed, elective surgery should be postponed for at least 1 month after all liver function tests return to normal values. If surgery is

Table 3. *Clinical and Laboratory Classification of Patients with Cirrhosis in Terms of Hepatic Functional Reserve by Child*

GROUP DESIGNATION	"A" MINIMAL	"B" MODERATE	"C" ADVANCED
Serum bilirubin* (mg per cent)	Below 2.0	2.0 to 3.0	Over 3.0
Serum albumin (gm per cent)	Over 3.5	3.0 to 3.5	Under 3.0
Ascites	None	Easily controlled	Poorly controlled
Neurologic disorder	None	Minimal	Advanced, "coma"
Nutrition	Excellent	Good	Poor, "wasting"

*Equivocal in biliary cirrhosis.

required for a patient with chronic active hepatitis,[28] it is desirable that the disease be quiescent for at least 3 months prior to operative intervention. Bromsulphalein (BSP) test is useful in disclosing latent liver diseases, such as chronic active hepatitis in the quiescent period, cirrhosis, or fatty liver. BSP test is also recommended when the patient has a history of liver disease, or a suspicion of liver impairment. If BSP retention at 30 minutes exceeds 15 per cent, the patient should be considered to have a significant decrease in hepatic reserve and be treated as a cirrhotic. Moreover, patients with alcoholic hepatitis should have elective surgery postponed until maximum recovery of liver function is determined, which generally takes from 1 to 3 months. Determination of surgical risk for patients with cirrhosis is made by referring to Child's classification[11] of hepatic functional reserve (Table 3). Patients in class "A" are well compensated cirrhotics who can tolerate rather complicated surgery with minimum risk; those in class "C" who are decompensated cirrhotics may not tolerate even simple surgical procedures and will often develop severe hepatic insufficiencies postoperatively. Patients who continue to have prolonged prothrombin time (greater than 4 seconds above control) after a loading dose of vitamin K (50 to 75 mg intravenously) along with a serum albumin level of less than 3 gm per 100 ml, are considered to have marked liver dysfunction and are extremely poor surgical risks.

There are a number of specific postoperative complications which invariably occur to a greater or lesser degree in most patients with seriously depressed hepatic reserve.

Fluid, Electrolyte, and Nutritional Disorders

Postoperative fluid retention and ascites is usually a reflection of an exacerbation of underlying liver disease or is due to sodium and fluid

overload; or occasionally both. There are three major factors contributing to fluid retention: (1) derangement in Starling's equilibrium by portal hypertension and hypoalbuminemia, (2) physiologic and biochemical changes in endocrine metabolism and renal function, and (3) a speculative unknown "third factor."[6]

In cirrhotic patients, particularly those with ascites, total body water and exchangeable sodium[5] are remarkably increased as is the plasma volume.[43] Cirrhotic patients frequently excrete less than 10 mEq of sodium per day regardless of sodium intake. A resultant water retention occurs to maintain isotonicity by way of renal osmoregulatory mechanisms. Glomerular filtration rate[3] is often reduced in cirrhotic patients; it is profound in those with intractable ascites. Consequently, the quantity of solute available for excretion with water in the kidney is limited. Secondary hyperaldosteronism[79] plays a major role in sodium retention in these patients. The diseased liver fails to inactivate aldosterone metabolically on the one hand, and reduced renal perfusion stimulates excess synthesis on the other. The plasma concentrations of renin, angiotensin, and angiotensinase are elevated in cirrhotic patients by the same mechanisms. Mineralocorticoid hormones,[48] estrogens, and vasopressin[58] also contribute[41] to fluid retention similarly.

Hyponatremia[29] is invariably a dilutional phenomenon in cirrhotic patients. This process should be treated by fluid restriction rather than by infusion of saline. Potassium deficiency[29] is also common in patients with liver disease. While chronic diuretic therapy contributes to hypokalemia, the magnitude of potassium deficiency has been shown to parallel the degree of liver dysfunction independent of diuretic therapy. Secondary hyperaldosteronism, renal tubular acidosis, and intravenous glucose solution all contribute to hypokalemia. Potassium supplementation in these patients is often necessary, but must be performed judicially since abrupt hyperkalemia may ensue if progressive renal failure occurs.

Magnesium deficiency is a common finding in patients with chronic liver disease. Symptoms are usually nonspecific; confusion and coma are the common findings in severe protracted cases. Zinc deficiency is particularly common in the alcoholic with cirrhosis resulting in suppression of wound healing[42] and immunity[17, 36] as is true for other nutritional deficits in these patients. Magnesium sulfate and zinc sulfate or oxide may be administered in severe cases.

The total body weight, fluid intake and output, caloric intake, serum electrolytes, and renal function tests must be frequently determined along with sequential liver function tests before and after major surgery. Fluid and sodium retention is prevented by restrictions. Adjunctive therapy in protracted cases includes diuretics and intravenous human albumin solution. Nutritional status of the patient may be improved preoperatively by using high calorie, high protein, and high vitamin diet. Postoperatively, the diet should be resumed as soon as possible. If adequate caloric intake cannot be established, intravenous parenteral nutrition should be started immediately.

Postoperative Bleeding Diatheses

The patient with liver disease may exhibit a bleeding tendency since the liver synthesizes many coagulation factors. Vitamin K dependent coagulation factors synthesized in the liver are factors II, VII, IX, and X. Pronounced deficiency of these factors occurs in patients with long-standing, deep jaundice and in patients who have received a prolonged course of oral antibiotics (Neomycin).

Hypofibrinogenemia of moderate degree is occasionally found in acute and chronic liver disease and usually suggests a poor prognosis. Hypofibrinogenemia may result from impaired synthesis of fibrinogen, from its rapid consumption during an intravascular coagulopathy, from its loss due to severe hemorrhage, or from its increased destruction by increased serum plasmin activity. Serum plasmin activity is invariably increased in liver disease. Most often, treatment of profuse postoperative bleeding with fresh frozen plasma will correct hypofibrinogenemia and other abnormalities. Occasionally, isolated fibrinogen can be given although it carries with it a significant risk of viral hepatitis. For intractable bleeding, epsilonamino-caproic acid is used to correct the elevated plasmin activity.[26]

Severe, protracted postoperative bleeding will invariably be associated with profound thrombocytopenia. Occasionally, platelet transfusions are necessary as an adjunct to cessation of bleeding in these cases. Lastly, in fulminating hepatic failure, a diffuse intravascular coagulation disorder may occur due to the release of tissue thromboplastin from disrupted hepatocytes. Under these circumstances, fresh frozen plasma and platelet transfusions may not be effective. Heparinization should be considered to prevent further consumption.

Postoperative Neuropsychiatric Disorders

Postoperative hepatic encephalopathy is usually accompanied by deteriorating liver function and is precipitated by events such as infection, gastrointestinal hemorrhage, constipation, electrolyte disorders (magnesium or potassium deficiency), hypoglycemia, or certain medications such as narcotics, analgesics, sedatives, hypnotics, and diuretics. These agents should be used judicially or meticulously avoided in patients with liver disease. The typical clinical picture is increased drowsiness, leading to somnolence and coma. The patient may have hepatic fetor, asterixis, and rigidity of the limbs. Slow rhythm on EEG, although not diagnostic for hepatic encephalopathy, correlates well. Hepatic coma must be differentiated from delirium tremens, particularly if the patient is a known alcoholic. The clinical picture of delirium tremens is usually characterized by terrifying hallucinations, fine tremor, tachycardia, sweating, and noisy delirium. The EEG does not show the classic slowing as seen in hepatic encephalopathy. Moreover, postoperative psychosis may mimic hepatic encephalopathy. In postoperative psychosis, there are usually no significant neurologic signs and the EEG remains normal.

Early diagnosis of true hepatic encephalopathy is extremely important in order to withdraw pharmacologic agents which may be causative mechanisms and to institute appropriate treatment. Whenever hepatic encephalopathy is suspected a strict regimen of protein restriction, sterilization of the bowel by neomycin, and cleansing of the gastrointestinal tract should be instituted.

Concomitantly, a search for other precipitating causes such as magnesium and potassium deficiency, gastrointestinal bleeding, and occult sepsis must be initiated and appropriately treated. Hepatic coma due to severe liver failure is most difficult to treat once it is fully established. Exchange transfusions,[61] hemodialysis,[39] charcoal hemoperfusion,[10, 56] and perfusion[23] through homologous or heterologous livers[1, 67] have all been used with occasional success. Presently, it appears that the only chance for long-term survival in these patients will be further development of liver homotransplantation; the 1-year survival in liver transplantation being approximately 35 per cent.[70]

Cardiorespiratory Complications

Characteristic alterations in cardiopulmonary dynamics occur in patients with liver disease. Arterial oxyhemoglobin saturations and oxygen tension are frequently low primarily due to intrapulmonary arteriovenous shunting and due to communications between the portal venous system and the pulmonary venous system. Cardiac output is increased as high as 15 liters per minute[37] due to decreased peripheral arterial resistance caused by extensive arteriovenous shunting. When the stress of major surgery is added to an already volume-overloaded heart, cardiac failure may develop. Notably, patients with cirrhosis may also have primary myocardial disease which further aggravates cardiac failure. Prophylactic digitalization and use of diuretics are appropriate in order to decrease the load on the failing heart.

Postoperative atelectasis and pneumonia are frequent complications in patients with chronic liver disease. These events are due to a number of causes including generalized weakness, poor nutrition, pulmonary A-V shunting, and often elevated diaphragm in patients with ascites. Frequently, respiratory support is required for intervals after general anesthetics are used.

Renal Failure

Five distinct types of renal failure have been known to occur postoperatively in patients with hepatobiliary disease: (1) acute tubular necrosis due to circulatory failure (i.e., cardiac failure, hypovolemia, sepsis), (2) drug nephrotoxicity (aminoglycosides, amphotericin B, etc.), (3) postoperative renal failure in patients with obstructive jaundice,[13, 14] (4) the "hepatorenal syndrome" of undetermined etiology,[12, 52] and (5) acute renal failure following hepatic artery ligation for hepatic tumors.[35] The first two (acute tubular necrosis and drug nephrotoxicity) will be

discussed in another article. Moreover, hepatic artery ligation causing acute renal failure is discussed in detail under the section involving complications of hepatic artery ligation. The remaining two types of renal failure will be discussed at this time.

Postoperative Renal Failure in Obstructive Jaundice

Many investigators have reported a high incidence of renal failure following operations on deeply jaundiced patients[13, 14] as compared to low incidence after comparable operations on patients who are not jaundiced. The high serum levels of bilirubin or bile salt are postulated as being responsible for the toxic effect on the renal tubules. Prophylactic treatment to avoid this form of renal failure appears to decrease its incidence significantly. Treatment includes intravenous hydration preoperatively, intraoperatively, and postoperatively with crystalloid fluids and adjunctive diuretics, usually mannitol.[13] It is particularly important to avoid renal failure in this group of patients since it carries a mortality of approximately 70 per cent.

"Hepatorenal Syndrome"

Oliguric renal failure occasionally occurs in advanced stages of liver disease. The term "hepatorenal syndrome" which was first used to describe postoperative renal failure in patients with obstructive jaundice has been used nonspecifically to describe any disorder with both hepatic and renal components. In fact, hepatorenal syndrome is a very specific entity which can only be diagnosed after first excluding the other common causes of acute renal failure in patients with liver disease.[12] Cardiac failure, hypovolemia secondary to hemorrhage, and sepsis are all common causes of acute renal failure (acute tubular necrosis) in patients with liver disease. Further, endogenous as well as exogenous toxins may cause nephrotoxicity and acute renal failure. Lastly, patients may develop renal failure of organic etiology independent of the liver disease. None of these etiologies represent the true "hepatorenal syndrome."

A select group of patients with liver disease develops oliguric renal failure without an identifiable cause. This is the "hepatorenal syndrome" in the modern sense.[12, 52] It is a functional form of renal failure without morphologic alterations in the renal structure. Absence of organic kidney disease in these patients has been documented clinically in two ways. First, patients with "hepatorenal syndrome" have donated their kidneys for a renal transplantation and the kidneys functioned immediately with normal renal function.[40] Contrariwise, patients with "hepatorenal syndrome" and overt liver failure have undergone liver transplantation. In the presence of an abrupt improvement of liver function postoperatively, the kidney function returns to normal.[33] The only abnormal parameters noted in kidneys experiencing "hepatorenal syndrome" are a marked decrease in renal cortical flow with a well maintained medullary flow. The mechanism postulated is cortical vessel constriction due to increased levels of serum angiotensin or by deficiency of prostaglandins. Suffice it to say that the diagnosis of "hepatorenal

Table 4. *Differentiation Between "Hepatorenal Syndrome" and Renal Tubular Necrosis*

	HEPATORENAL SYNDROME	RENAL TUBULAR NECROSIS
Ascites	Usually	Possible
Hepatic function	Poor	Variable
Precipitating factor	Possible	Yes
Oliguria	Late	Early
Urine specific gravity	>1.010	±1.010
Urinalysis	Normal	Abnormal
Tubular Na reabsorption	Increased	Decreased
Tubular K reabsorption	< Na	> Na
Osmolality of urine	High (> plasma)	Low (< plasma)
Specific renal histology	No	Yes

syndrome" is made by exclusion of other causative mechanisms. The characteristic laboratory findings are: (1) capability of the kidney to concentrate urine (specific gravity usually 1.015 or greater), (2) normal or nonspecific urinalysis, and (3) extremely low urinary sodium excretion (10 mEq per day or less) (Table 4).

There is very little to offer in the way of treatment for this syndrome other than conventional treatment of hepatic failure and renal failure.[12] Overt hepatic and renal failure due to hepatorenal syndrome invariably results in death, even in the presence of aggressive hemodialysis. Patients invariably die of hepatic failure, complications of hepatic failure, or those of renal failure. At present, hepatic homotransplantation is probably the only treatment modality which offers a reasonable opportunity for survival.

SPECIFIC COMPLICATIONS IN SURGERY OF THE LIVER

Complications of Hepatic Resection

With an increased understanding of anatomy and physiology of the liver along with modern management of the patient, major hepatic resections can now be performed with an acceptable risk. The specific anatomic technical details involved in hepatic resections are beyond the scope of this article and we refer the reader to the articles of Starzl,[69] Lin,[46] and Fortner.[19] A detailed knowledge of the anatomy of the liver is essential in hepatic resection. Preoperative angiographic studies (both celiac and superior mesenteric angiography) provide a helpful map to guide the performance of major hepatic resections safely.

Avoidance of Intraoperative Complications

Hepatic Artery and Portal Vein. Since the liver receives approximately 3 liters of blood per minute, the technical success during resection depends totally upon hemostasis. Arterial and portal venous bleeding along the surface of the hepatic resection are rather easily

avoided by first ligating the major extrahepatic portal vein and hepatic artery to the lobe to be resected. Further, excess blood loss may be avoided by intermittently compressing the extrahepatic portion of each vessel in the porta hepatis. If blood loss is accurately measured and adequately replaced by transfusions during operations, serious hypovolemic episodes do not occur. However, even a modest amount of blood loss can lead to cardiac arrest if compression or torsion of the hepatic portion of the vena cava occurs during resection, resulting in blockage of venous return from the lower half of the body. Lifting and manipulation of the liver should therefore be performed carefully so as not to obstruct the vena cava. Constant communication with the anesthesiologist during the manipulation of the liver invariably averts this catastrophic accident.

HEPATIC VENOUS BLEEDING AND AIR EMBOLI. Uncontrollable, life-threatening bleeding is usually due to inadvertent injury to hepatic veins and/or intrahepatic vena cava during dissection. Special attention should be exercised not to injure the caval wall while ligating the hepatic veins behind the liver. Wide exposure of both the suprahepatic and infrahepatic cava should be performed in order to control bleeding should it occur. Positive end-expiratory pressure should be applied during this procedure to prevent air emboli. If the caval wall is inadvertently torn and the bleeding is not easily controlled, the area should be packed and gentle pressure applied. The anesthesiologist should then be instructed to transfuse the patient to an elevated central venous pressure since there is likelihood that the inferior vena cava may require cross-clamping. Once the patient has an expanded intravascular volume, the packing should slowly be removed in order to identify the injury. If the injury is not easily repaired, a variety of techniques may be used. First a Foley catheter may be inserted into the hole and subsequently inflated. Gentle traction on the catheter will occlude the injured area and closure of the injury is performed over the balloon. Prior to approximation of the last ligatures, the catheter is decompressed and removed. If this procedure is untenable, the vena cava should be cross-clamped above and below the liver and at the same time the common hepatic artery and portal vein should be compressed. The liver will tolerate approximately 15 minutes of warm ischemia. If the injury cannot be repaired within this time limit, cooling of the liver is necessary. The hepatic side of the portal vein is cannulated and perfused with 2000 ml of cold heparinized lactated Ringer's solution (6° C.) followed by cannulation of the gastroduodenal artery and infusion of 200 ml of the same solution into this branch of the common hepatic artery. Once the liver is chilled, it will tolerate anoxia for 1 to 2 hours safely. If a large hepatic tumor is resting on the hepatic portion of the vena cava, the technique of vascular isolation and hypothermic perfusion described by Fortner[19] can often be lifesaving.

HEPATIC VEIN OUTFLOW BLOCK. Uncontrollable profuse bleeding may occur from the cut surface of the nonresected lobe if the major hepatic vein draining this remaining lobe has been accidentally strictured or ligated and no other hepatic veins remain sufficient to drain the venous blood. For example, the middle hepatic vein quite often joins the

left hepatic vein and inadvertent ligation or stricture of this latter vein during extended right hepatectomy or trisegmentectomy creates a striking outflow block as in a Budd-Chiari syndrome. This abrupt hepatic venous hypertension results in diffuse and massive bleeding from the cut surface. If profuse bleeding occurs, it is mandatory to re-evaluate integrity of the remaining major hepatic vein before considering a coagulation disorder as the cause. Removal of the ligature or alternately hepatic vein-vena cava bypass is mandatory to prevent exsanguination and/or liver failure.

Postoperative Complications

Bleeding. Even after most careful hemostasis is confirmed before closure, re-exploration within 24 hours of hepatic resection is not a rare event. A coagulation profile should be determined at the end of surgery and abnormalities should be corrected immediately. If bleeding continues in spite of adequate replacement therapy, as discussed earlier, re-exploration should not be delayed. The following areas must always be thoroughly examined: (1) cut surface, (2) vena cava, (3) inferior phrenic veins, (4) lumbar veins, (5) adrenal gland, (6) hepatic hilum, (7) duodenal vessels, and (8) the spleen.

Acute Hepatic Failure. Fulminant acute hepatic failure is a rare complication since the normal liver has an extreme reserve capacity. This complication is usually fatal, and most often occurs after either: (1) extensive resection of a cirrhotic liver, (2) accidental ligation of the common hepatic artery, portal vein, or common bile duct, or (3) systemic sepsis. Careful surgical technique, meticulous patient care, and prudent treatment of the cirrhotic patient will help to avoid this fatal complication. Less severe hepatic failure is not infrequent following intra- and postoperative hypotension associated with prolonged hypoperfusion of the liver during resection. Integrity of liver function usually improves with medical support, unless progressive renal failure or sepsis develops.

Biliary Fistula. A modest amount of bile stained drainage for a few postoperative days is usual and is most often not significant. A large amount of bile drainage for a prolonged period is usually a result of unnoticed injury to the common bile duct or accidental stricture or ligation of the bile duct. Routine use of a T-tube to drain the common bile duct is no longer recommended[20, 48] since invasion of a normal size common bile duct may result in stenosis or ascending cholangitis. Further, decompression of a nonobstructed biliary tree will not decrease bile leakage from the cut surface. Intraoperative cholangiogram should be obtained, before and after hepatic resection. Cholangiography can be easily performed through the cystic duct for the left lobectomy. Intrahepatic bile ducts are clearly visualized on the x-ray films, when the abundant radiopaque dye is injected, with the common duct being compressed by fingers. Cholangiograms before the resection may disclose aberrant branching of the bile duct and those after resection disclose the presence or absence of bile duct injury or bile leakage. Cholangiographic evidence of integrity of the biliary tree after resection provides reassurance that persistent biliary leak and/or postoperative jaundice

are not due to biliary obstruction. Adequately drained bile fistula from the cut surface in the absence of bile duct obstruction will eventually cease within 60 days.

POSTOPERATIVE JAUNDICE. Postoperative jaundice is quite common after major hepatic resection. Bilirubinemia is most often due to the insult of major hepatic resection and the effect of multiple transfusions. Bilirubin gradually increases for a few to several postoperative days and begins to subside after 5 to 10 days. Serum bilirubin levels of 10 mg per 100 ml or greater are not uncommon. Serum alkaline phosphatase also increases from two to three times normal levels for 10 to 14 days. The differential diagnosis may be extremely difficult, but unnecessary re-exploration should be avoided using various diagnostic procedures. Certainly, intraoperative cholangiography provides presumptive evidence of a nonobstructive etiology. The details involving the differential diagnosis of obstructive jaundice have been discussed earlier.

SUBPHRENIC ABSCESS. Subphrenic abscess is probably the most common late postoperative complication after hepatic resection. Since inadequately drained exudate, blood, bile, lymph, and devitalized tissue are the causes of infection, the surgical area should be left extremely dry before the closure. Moreover, meticulous attention to adequate drainage at the time of operation is a necessity. Pendular drainage of the entire perihepatic space with as many as ten to twelve Penrose drains is necessary and should be accompanied by sump drains using continuous low suction, draining the most dependent area of the upper abdomen. Drains should usually exit through a generous (5 cm) incision near the tip of the 12th rib on the side of the resection.

Prolonged fever and leukocytosis usually associated with low-grade jaundice and local tenderness signify subphrenic abscess. Chest x-ray films often delineate an elevated hemidiaphragm and occasionally pleural effusion. Sonogram or gallium scan is helpful to confirm the diagnosis, but re-exploration and drainage through a generous incision is mandatory. Use of a sump-suction drain and Penrose drains provides the most adequate form of drainage. We personally add a peritoneal catheter to the involved area and lavage with an appropriate antibiotic solution for 5 to 10 days.

ASCITES AND EDEMA. Ascites and edema result from decreased albumin synthesis and quite often from a mild degree of portal hypertension. Hypoalbuminemia may last 2 to 4 weeks postoperatively and daily intravenous administration of 25 to 75 grams of human albumin along with doses of diuretics and sodium restriction are useful for short periods of time.

POTASSIUM, PHOSPHORUS, AND GLUCOSE DERANGEMENT. Severe hypokalemia and hypophosphatemia may occur after major hepatic resection, particularly if a large amount of stored red cells are transfused. Hypoglycemia, as well as hyperglycemia, is also frequent. Stored red cells are depleted in potassium and phosphorus as are the hepatocytes after operative injury. Potassium and phosphorus influx into the injured cells and consequently deplete extracellular concentrations of these ions occasionally to critical levels. Further, high concentrations of

glucose solution, usually administered after hepatic surgery, shifts serum potassium and phosphorus into cells. Hypoglycemia may occur when hepatic glycogen stores are depleted. In contrast, if glycogenesis is severely impaired by hepatic surgery, extensive amounts of glucose infusion may lead to hyperglycemia. Therefore, serum glucose and electrolytes should be monitored closely intra- and postoperatively. Depletion of potassium and phosphorus should be treated with replacement therapy: hypoglycemia with hypertonic glucose, hyperglycemia with insulin.

COMPLICATIONS OF HEPATIC ARTERY LIGATION

The normal liver receives 70 to 80 per cent of its blood supply from the portal vein and the remainder from the hepatic artery. The portal vein blood is rich in oxygen and probably up to 70 per cent of oxygen to the normal liver is supplied through the portal vein. For more than a decade, hepatic artery ligation has been used for nonresectable malignant tumors of the liver with promising results.[18, 55] Later, this treatment was applied for hepatic trauma[50] and for hemobilia in order to accomplish hemostasis. Ligation of a lobar artery (right or left) is usually well tolerated and is all that should be performed when the injury is confined to one lobe. Ligation of the hepatic artery propria (distal to the gastroduodenal branch) is more hazardous since it reduces the chance for collateral flow from the other lobe. Note that hepatic artery ligation should not be routinely applied for hemostasis unless the bleeding is uncontrollable and its arterial origin is confirmed by intermittent arterial compression. Unnecessary hypoxia to the traumatized liver may be fatal. The normal human liver will tolerate hepatic artery ligation for the following two reasons: (1) the portal venous blood, rich in oxygen, can supply enough oxygen to the liver to maintain metabolic integrity, and (2) collateral arteries in the intact lobe, falciform ligament, hepatic hilum, and diaphragm develop and supply enough oxygen to the liver within a few days.

Prior to ligation of the hepatic artery for devascularization of either primary or secondary hepatic tumors, a celiac and superior mesenteric arteriogram is essential. The arteriogram will not only determine anatomic variations of the hepatic blood supply, but will also delineate the highly vascularized tumor which is most likely to respond to this treatment modality. The following relative indications should be considered to avoid serious or fatal complications: (1) the patient should be relatively active and not be confined to bed, (2) the patient should not be clinically jaundiced and should not have a significant amount of ascitic fluid, (3) the liver is not cirrhotic, (4) the portal vein is patent and perfusing the liver, (5) at least 30 to 40 per cent of normal liver tissue is left intact, (6) renal function is normal, and (7) no intercurrent infections are present in the patient.

Postoperative Bleeding

Postoperative bleeding is not a rare complication after hepatic artery ligation. The liver is usually quite enlarged because of tumor or tumors and therefore extensive dissection around the hepatic hilum is usually necessary to locate the artery. Further, acute ischemic insult to the liver usually results in a transient coagulopathy due to depletion of the liver-based clotting factors. For these reasons, meticulous hemostasis is a necessary part of this operation. Moreover, if a catheter is inserted into the artery for later chemotherapy, it may become dislodged and cause massive postoperative hemorrhage.

Hepatic Failure

Ischemic hepatic injury is unavoidable after hepatic artery ligation. All liver function tests including a coagulation profile become transiently abnormal. Serum transaminase levels and LDH usually reach 1000 to 10,000 units within 24 hours and rapidly decline to 200 units in 7 to 10 days. Alkaline phosphatase rises sharply for the first few days. Serum bilirubin gradually increases but does not usually exceed 5 mg per 100 ml if hepatic failure does not ensue. When hepatic failure does occur, it appears gradually and progresses to overt hepatic failure within weeks. In spite of intensive medical management, this complication is usually fatal. While the response to hepatic artery ligation is unpredictable, the relative indications previously mentioned represent rough guidelines to be used to predict whether the liver will tolerate this acute ischemic insult.

Acute Renal Failure

Acute renal failure which precedes clinical hepatic failure after hepatic artery ligation is probably a specific complication occurring in patients with bulky, very vascular, malignant tumors.[35] Renal failure develops quite suddenly within 24 hours after ligation and does not seem to be secondary to hypotension or hypovolemia. Urine is concentrated and high in sodium concentration. These values mitigate against acute tubular necrosis or hepatorenal syndrome as a diagnosis. Concurrently, marked elevation of transaminases, lactic dehydrogenase, and uric acid occur. Serum potassium increases rapidly with profound oliguria. These findings strongly indicate massive necrosis of both the tumor and the liver as has been verified at postmortem examination studies. It has been postulated that release of toxic substances from the necrotic tumor causes acute renal failure. Pre- and postoperative hydration as well as infusion of mannitol for the first 24 to 48 hours after ligation appears to provide prophylaxis for this type of acute renal failure – presumably due to rapid renal excretion of toxic products from necrotized tumors.

Sepsis

Sepsis is the third serious complication after hepatic artery ligation. Mild to moderate degrees of fever and leukocytosis are common after hepatic artery ligation, probably due to tumor necrosis. Necrotized tumors are ideal culture media for bacterial growth. Intraoperative bac-

terial contamination, infection through drains, and contaminated arterial catheters for chemotherapy all contribute to hepatic sepsis. Therefore, postoperative blood cultures should be obtained routinely. Pre-existing infections must be eradicated before hepatic artery ligation is to be performed; prophylactic antibiotics should be started preoperatively and continued for 3 days.

Candidates for hepatic artery ligation for the most part have advanced malignant tumors and are usually nutritionally debilitated. Close monitoring and intensive medical support are the keys to successful surgery. These patients should be prophylactically treated for impending hepatic and renal failure. Electrolyte balance, nutrition, renal and hepatic support, as well as prophylaxis for infection, are mandatory.

COMPLICATIONS OF PORTAL DECOMPRESSION SURGERY

Since Whipple and Blakemore successfully introduced portacaval shunt for the treatment of portal hypertension in 1945, well over 100,000 portasystemic shunts have been performed. Standard end-to-side and side-to-side portacaval shunt, standard and H-graft mesocaval shunts,[15, 47, 72] and standard splenorenal shunts have been used to decompress the portal system. Each shunt has its own technical advantages and disadvantages depending on the individual anatomy. In general, the standard end-to-side portacaval shunt is the easiest to perform and is least likely to be clotted. Standard side-to-side shunt is sometimes difficult to perform when the caudate lobe of the liver is enlarged. The standard splenorenal shunt is often thrombosed if the diameter of the splenic vein is less than 10 mm. In cases of portal vein thrombosis, the splenorenal shunt or mesocaval shunt is necessarily the operation of choice. The H-graft mesocaval shunt provides a safe method for constructing a side-to-side shunt when the portal vein is inaccessible either due to previous surgery or due to an enlarged caudate lobe.

Although portal decompression surgery is the most effective treatment of bleeding esophageal varices, enthusiasm has declined because of postshunt encephalopathy[54] and due to unfavorable results in control studies of prophylactic shunts. Nevertheless, portasystemic shunts continue to be the primary treatment for bleeding esophageal varices.[53, 62] Three major complications after portal decompression occur: (1) recurrent gastrointestinal bleeding, (2) postshunt encephalopathy, and (3) postshunt ascites.

Recurrent Gastrointestinal Bleeding

Gastrointestinal bleeding after portal systemic shunting is not an infrequent event. It may result from ruptured esophageal varices, alcoholic gastritis, peptic ulcers, or Mallory-Weiss syndrome. Endoscopic examination is used to determine the source of bleeding and provides a diagnosis for appropriate management. Variceal bleeding after shunt operations is most commonly due to thrombosis of the anastomosis. In order to confirm thrombosis of the shunt, splenoportography and central

venous catheterization are quite helpful. High-splenic pulp pressure and nonvisualization of the portal systemic shunt are conclusive for the diagnosis of the clotted shunt. Placement of a catheter tip through the anastomosis under fluoroscopic control invariably documents patency of the shunt. If the catheter tip fails to pass the anastomosis, blood samples for ammonia and glucose levels should be taken both proximal and distal to the anastomosis. Since portal blood has higher concentrated ammonia and glucose than systemic blood, the proximal blood samples should have appropriately elevated values for these substances if the shunt is patent. The absence of elevated levels indicate a thrombosed shunt. When a patient experiences variceal bleeding due to a thrombosed shunt, either a secondary portal systemic shunt or an ablative surgery for esophageal varices should be performed to prevent further hemorrhage.

Postshunt Encephalopathy (Eck's Syndrome)

Hepatic encephalopathy after a portal systemic shunt operation is well known as Eck's syndrome. Severe postshunt encephalopathy occurs in 20 to 40 per cent of alcoholic cirrhotics, and even more frequently in patients with postnecrotic or cryptogenic cirrhosis. Factors leading to postshunt encephalopathy include: (1) impaired hepatic function due to decreased hepatic blood flow, (2) shunting the portal blood, rich in encephalogenic substances which have not been detoxified by the liver, and (3) increased absorption of encephalogenic substances from the decongested intestines.[31]

The treatment of postshunt encephalopathy[65] is primarily medical supportive management and has been discussed in an earlier portion of this section dealing with hepatic encephalopathy. Restrictions of protein intake, enhancing bowel movements, and the sterilization of the bowel most often improve encephalopathy by reducing the production of and the intestinal absorption of encephalogenic substances.

If encephalopathy is persistent and severe, resulting in debilitation of the patient, surgical intervention may become indicated. Total colectomy,[2] exclusion of the colon by ileosigmoidostomy, or even closure of the portal systemic shunt[34, 51, 63] may become necessary on some occasions. A selective shunt (distal splenorenal anastomosis,[22, 77] gastric coronary vein–caval anastomosis or ablative surgery[81]) for varices should be performed at the time of the shunt closure to avoid risking subsequent hemorrhage from esophageal varices.

Disparate opinion persists as to the physiologic advantage of the various sites of portal systemic shunts. All types of portal systemic shunts which decompress the portal system can be hemodynamically classified into either end-to-side or side-to-side portacaval shunt depending on the continuity of the portal vein. Since standard splenorenal, mesocaval, and H-graft mesocaval shunts are all minor modifications of the standard side-to-side portacaval shunt, none of these has yet been proved to be significantly superior to the standard side-to-side portacaval shunt. Reliable clinical data comparing end-to-side and side-to-side portacaval shunts are sparse,[8, 32, 57] but strongly suggest that there is probably no remarkable difference in the incidence of postshunt encephalop-

athy between these two types of standard portacaval shunts.[74, 82] The data reporting alterations in the hepatic blood flow are controversial depending upon the method of measurement. Indirect measurements of hepatic blood flow (estimated hepatic blood flow) by hepatic excretion of dye have delineated a lesser decrease in blood flow after the end-to-side shunt,[59, 64] but direct measurement by electromagnetic flowmeters have revealed less decrease after side-to-side shunts.[8, 57]

Postshunt Ascites

Ascitic fluid of cirrhosis is a transudate from the liver surface and is caused by increased postsinusoidal resistance to hepatic blood flow. The transudate cannot be efficiently reabsorbed by the peritoneum because of high portal pressure and low serum oncotic pressure. Both end-to-side and side-to-side portal systemic shunts are most often effective in controlling cirrhotic ascites. However, potent diuretics are also effective in controlling ascites in most patients, thus precluding the frequent use of portal systemic shunts for ascites. Further, ascites which is refractory to diuretic therapy has recently been effectively treated using the LeVeen shunt.[43]

Refractory ascites has occasionally become a major clinical problem even after a successful portal systemic shunt—particularly of end-to-side type. If the hepatic postsinusoidal pressure is great enough to reverse the direction of portal blood flow, then an end-to-side portacaval shunt will not be decompressing the sinusoids and may even increase the sinusoidal pressure by interrupting the retrograde hepatic flow of blood through the portal vein. As a result of increased hepatic sinusoidal pressure, the rate of formation of ascitic fluid may actually increase after construction of the shunt. In order to avoid development of postshunt ascites, measurement of the occluded portal vein pressure must be performed intraoperatively. When the occluded portal vein pressure[76] on the hepatic side is higher than the nonoccluded pressure, a side-to-side type of portal systemic shunt is recommended rather than an end-to-side portacaval shunt. After side-to-side portal systemic shunt, the portal vein will serve as a hepatic outflow tract to decompress the sinusoidal pressure if the postsinusoidal resistance is elevated.

Postshunt ascites must be considered as a diagnostic possibility when severe ascites occurs after an end-to-side portal systemic shunt. Other common possibilities include thrombosed shunt, operative injuries to major lymphatics, peritonitis, and pancreatitis. Diagnostic paracentesis and angiography will provide a diagnosis. Treatment of postshunt ascites must first be aggressive use of diuretics. Refractory postshunt ascites should be treated with a LeVeen peritoneal-jugular shunt.

SUMMARY

Hepatic dysfunction is one of the most common complications following major surgical intervention, and it presents a wide spectrum of clinical manifestations as discussed. Recent progress in hepatology has

provided better knowledge in etiology and pathophysiology of hepatic dysfunction and its sequelae. In addition, modern medical technology has made the diagnosis of liver disease easier and more precise. Underlying liver disease should be disclosed precisely prior to surgery and surgical complication minimized for patients with hepatic impairment. If hepatic complications occur following surgical intervention, the most logical etiology and pathophysiologic explanation should be found, utilizing all the clinical and laboratory data and diagnostic procedures in order to institute proper treatment.

Specific complications in hepatic surgery are also discussed. A good understanding of pathophysiologic alterations following hepatic surgery is mandatory together with detailed anatomic knowledge and surgical skill in order to prevent and to treat serious complications.

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Head and Neck Surgery

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Most complications in head and neck surgery can be defined in terms of blood vessel and nerve involvement. It is from this perspective that the complications in surgery in and about the mouth and pharynx, parotid region, and neck (exclusive of the thyroid region, which is dealt with in another article in this symposium) will be discussed.

VASCULAR SYSTEM

Arteries

Postoperative complications of necrosis of the skin, mucosa, muscle, and perhaps occasionally nerve, bone, and cartilage are due to the cessation of blood flow, particularly through arterial interruption. Though venous flow must also be adequate to maintain the viability of tissues, the anatomic predisposition for adequate collateral runoff is much greater than the arterial collaterals and is therefore less critical in most clinical situations. Success in maintaining viable tissues after surgery is dependent upon retaining adequate blood flow to the tissues from collateral sources when arteries to a part or parts must be interrupted. The knowledge of collateral arterialization to the parts of the head and neck, as well as the functional effects of partial arterial interruption builds the basis for proper incision and surgical dissection and therefore the avoidance of many complications.

Clinical vascular consideration will be discussed by tissues: brain, muscle, and skin.

BRAIN. Through the circle of Willis, one carotid artery is usually successful in supplying the contralateral area of the brain when a carotid artery is ligated. Unilateral ligation of the common carotid artery results in significant neurologic sequelae in less than 5 per cent of normovolemic and normotensive patients. This contrasts with a very significant neurologic morbidity and mortality of greater than 90 per cent in

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exsanguinating carotid wounds of battle casualties and of postoperative carotid artery blowouts following radical neck dissection. The variable between the elective and emergency situations is *blood volume*. It seems reasonable that when presented with exsanguinating hemorrhage from a carotid artery that the surgeon after tamponading the rent, first rapidly restore blood volume, if necessary, with type-specific uncrossmatched blood. Formal repair of the arterial wound should await the restoration of the blood volume and consequently the systemic and cerebral arterial pressure.

Fixation of a malignant tumor to the carotid artery is no longer an absolute contraindication to extirpative tumor surgery of the neck. When one anticipates the necessity for removal of the common and/or internal carotid system, one may test the collateral flow of the brain by temporary interruption of arterial flow under local anesthesia, while clinically evaluating the patient's mental status and voluntary movements.[3] Additional laboratory data in the form of brain wave recording (EEG) and blood pressure and flow studies can be used to evaluate the functional adequacy of collateral blood flow.[4] In those patients whose physiologic data imply a strong risk of a stroke, bypass surgery remains an alternative when carotid artery interruption is necessary. Uncommonly, temporary or permanent neurologic deficit can evolve over several days following ligation and this risk must be weighed against the benefits of the procedure. When one is faced with the necessity of interrupting the carotid arterial blood flow during the surgical procedure, it appears that a back pressure (readily measured) of 50 mm of mercury is adequate to maintain brain viability.[1]

Postoperative carotid artery blowout, though uncommon, remains a most significant postoperative complication of radical neck surgery. It is the result of necrosis of the arterial wall through loss of flow through the vasa vasorum. Contributing factors are: skeletonization of the artery during surgery, wound sepsis with direct enzymatic necrotizing effects of bacterial products upon the carotid wall, pressure necrotizing effects from hematoma and abscess formation, hypovascularization of the arterial wall due to irradiation effect. Skin necrosis occurs in the same manner as carotid arterial wall necrosis and is a concomitant and not a cause of carotid artery blowout unless the necrotic skin flap is allowed to contribute to uncontrolled wound sepsis.

Good surgical technique, with preservation (including prevention of desiccation of the arterial wall during surgery), will do much to prevent arterial wall necrosis. Careful and frequent postoperative wound observation with prompt treatment of wound infection and hematoma will likewise do much to prevent this disaster. A neck flap over the carotid arterial system that does not show the expected firmness of healing connective tissue organization in the first 4 to 5 days postoperatively or one in which pocketing of serosanguinous fluid occurs despite formal drainage should be investigated by adequate reopening of the wound in an appropriately dependent position. Through-and-through drains with regular and frequent saline irrigations or wide open packing and frequent dressing changes will prevent further necrosis of the areolar tissues and vasa vasorum of the carotid artery. The mere execution of carotid artery

coverage by levator scapulae muscle flaps and dermal grafts may give false security in the prevention of carotid artery blowout if careful surgical technique and optimal postoperative wound care is not carried out.

MUSCLE. Experimental studies by Le Gros Clark and Blomfield[5] have shown that ligature of a main artery at the site of entry into a muscle or interruption of a main intramuscular channel by incision of the muscle frequently leads to a sharply defined area of ischemia with ensuing necrosis. To some degree the inadequacy of intramuscular arterial anastomosis pertains to all muscles and also to those commonly manipulated in head and neck surgery. If the sternocleidomastoid muscle is mobilized from below upward, and if its main arterial input which enters the deep portion of that muscle in its upper half is transected during mobilization, a large portion of the sternocleidomastoid muscle will necrose. Likewise the levator scapulae muscle, which is used for flap coverage of the carotid artery in radical neck dissection, is easily dearterialized with subsequent necrosis when its arterial input into the upper half of this muscle is transected by overmobilization. Portions of the tongue, which is made up of several muscles covered by oral mucosa, may undergo ischemia and subsequent necrosis due to transection and ligature of a crucial set of arteries entering the tongue muscle. Where large portions of the base of the tongue are broadly skeletonized by efforts to widely excise malignant tumors and their node-bearing areas in the sublingual area, obliquely sectioned parts of the anterior two-thirds of the tongue may be dearterialized and will subsequently necrose. Such a remaining tongue muscle-mucosal flap when used in the oral closure of a surgical wound can only fail to heal and perhaps contribute to a septic orocervical wound.

When it becomes apparent during a surgical procedure that muscle tissue has been dearterialized and lacks a circulation, excision of the muscle should be carried back to viable tissue before the closure of the wound is initiated. If muscle necrosis is not detected until the postoperative period, debridement back to normally vascularized muscle should be carried out to control the sepsis within the wound.

SKIN. The skin of the neck receives its arterial supply from a subdermal plexus and this plexus in turn is fed by perforating arteries that pass through the investing cervical fascia at several points along the sternocleidomastoid muscle.[5] Arterial supply likewise passes from above downward at the lower border of the mandible and from below upward from below the clavicles into the skin of the neck. Ordinarily, wide dissection of skin flaps on the surface of the investing cervical fascia will provide adequately vascularized tissues for proper healing. Incisions in the neck which create acute angles tend to sequester skin points with a reduced arterial circulation. Though T incisions tend to hold up well under ideal circumstances of flap mobilization and closure, incisions which do not create angular flap tips (i.e., parallel transverse incisions) should be used when dealing with irradiated tissues or other circumstances that are less than ideal.

Exposure of the carotid sheath in elective surgery upon the carotid artery in the upper two-thirds of the neck is readily obtained through an

obliquely vertical incision along the anterior border of the sternocleidomastoid muscle. Skin flap mobilization, with its incumbent morbidity, is eliminated by this approach though transverse incisions usually leave a less apparent scar.

The same circumstances which mitigate against adequate blood flow to the carotid artery wall and cause necrosis apply in the case of skin flaps. In clinical terms this usually means undrained wound sepsis of hematoma and the management of this problem has already been discussed.

Mucocutaneous fistulas are the result of skin and mucosal necrosis. The immediate care of an incipient fistula is debridement of the necrotic tissues in the vicinity. If sufficient and mobile soft tissues are present in the area of the fistula and no tumor tissue or foreign bodies are present, the fistula will close spontaneously. When immobile, scarred, and hypovascular tissues (following irradiation) surround the area of fistula, satisfactory soft tissues must be mobilized to accommodate the skin and mucosal deficit. It is frequently necessary to widely excise the margins of the fistula to attain healing.

Veins

A rent in the internal jugular vein may be the source of rapid blood loss but also because of the negative pressure within the thoracic cage may be the source of air embolism. Ordinarily, obtaining hemostasis in injuries of the internal jugular vein in the base of the neck can be dealt with directly through the neck wound. However, the surgeon must have the facility for rapidly entering the chest when necessary to control a retracted stump of the internal jugular vein.

Uncontrollable bleeding at the exit of the internal jugular vein from the base of the skull which cannot be ligated can be packed. Troublesome bleeding from the fine plexus of veins that is found in and about the lateral pterygoid muscle in the infratemporal fossa is most often encountered in extended maxillary resections. This bleeding can most often be dealt with by ligature or electrocoagulation but on occasion packing, frequently over a perforated skin graft, is necessary.

Lymphatics

Injury to the large lymph ducts on either side of the neck in radical neck surgery is uncommon but increases as one carries a dissection nearer the junctions of the internal jugular and subclavian veins. When it is possible to detect an injury at the time of surgery, a suture ligature is placed at the site of the injury effectively preventing a postoperative lymphatic fistula. If a clear fluid rises in the depth of the wound during surgery, one must assume that the lymphatic duct has been injured. If necessary a microscope is used to detect the exact site of injury and fine suture ligatures are used to seal the opening. Due to the delta-like entry of the lymphatic ducts into the great veins of the base of the neck, ordinarily, only one of many trunks is injured and ligature of the single trunk does not impair the flow of lymph through the thoracic or right lymphatic ducts. If it appears that a lymph fistula exists, by the accumulation of a serum-like material in the wound drainage during the

postoperative period, then a waiting period of several weeks before attempts at formal repair is justified, as the great majority of lymph fistulas close spontaneously.

NERVOUS SYSTEM

Manipulation and contusion of a nerve can temporarily inhibit transmission with resulting lack of muscle or sensory function. If the surgeon does not visualize the entire course of an important nerve that passes through a surgical field, he is unable to determine if inadvertent transection of the nerve occurred during surgery when there is a sensory or motor loss detected postoperatively. In general if the entirety of the course of a nerve has been visualized in a given surgical wound, re-exploration would not be indicated when a functional loss occurs postoperatively, the expectation being that of spontaneous return of function. To the contrary, if such a deficit exists when the nerve has not been completely dissected, re-exploration is indicated if the deficit is important enough to be corrected.

The seventh, tenth, eleventh and twelfth cranial nerves are all encountered in surgery about the neck and parotid region. Permanent injury to all or part of these nerves varies from the mildest form of cosmetic deficit to a significant functional deficit.

The facial nerve is of enough functional and cosmetic significance to make it the primary landmark of dissections in surgery of and about the parotid gland. In parotidectomy the nerve is located near the exit from the stylomastoid foramen and its superficial surface is exposed up to the anterior, superior, and inferior margins of the parotid gland. This can cause a facial paralysis which may require 3 to 6 months for return of function. In total parotidectomy or secondary dissection within pre-existing scar where it is necessary to extensively skeletonize the finer branches of the facial nerve, such as that to the lower lip and the forehead, motor function may be permanently impaired, presumably due to both extra- and intraneural scar deposition.

In dissections of the upper neck near the angle of the mandible and those about the zygomatic arch and midcheek region, it is generally preferred to explore the wound to isolate the appropriate branches of the facial nerve. Once the nerve has been isolated it can be protected from injury. Landmarks for the isolation of the mandibular branch of the facial nerve in the neck have been reviewed by Dingman.[2]

The vagus nerve is important because of its sensory and motor innervation to the larynx. The superior laryngeal nerve which branches from the vagus near the base of the skull runs deep to the carotid arterial system and into the thyrohyoid membrane and is extremely important in the sensation of the larynx, preventing aspiration by appropriate motor reflexes. The bilateral loss of this nerve function makes it almost impossible for a patient to prevent aspiration and creates a most distressing situation. Ordinarily, this nerve is out of the operative field in most operations upon the neck with the exception of direct surgery for lesions of the larynx itself. The function of this nerve is extremely im-

portant in deglutition and all reasonable attempts at its preservation should be made. Inadvertent section of this nerve should be repaired by dissecting microscope, if and when necessary by nerve graft. The recurrent nerve is important to motor innervation of the larynx and will be dealt with in the section on thyroid and parathyroid complications.

The accessory nerve is motor to the sternocleidomastoid and trapezius muscles. The most common distressing long-term complication of radical neck dissection has been the arthritic symptoms of the shoulder referable to the section of the accessory nerve which eliminates the trapezius' function upon the shoulder. Some surgeons have modified their neck dissection techniques under certain clinical situations to exclude the transection of the accessory nerve. Caution must be maintained in any dissection in the posterior triangle of the neck in which purposeful section of the accessory nerve is not intended. Inadvertent section of the accessory nerve should be repaired as soon as possible by microscopic methods.

The hypoglossal nerve passes superficial to the carotid arterial system in the upper portion of the neck and is the sole innervation of half the tongue. Transection of this nerve, though causing atrophy of half the tongue, usually has little effect on tongue usage and is ordinarily well tolerated by a patient. On the other hand, it would appear appropriate that despite the low morbidity after transection of this large nerve, repair be carried out when inadvertent section of this nerve occurs at operation.

Transection of sensory and motor nerves from the cervical plexus are generally well tolerated and repair of those inadvertently sectioned are not normally repaired. An exception to this rule would be the phrenic nerve, the motor nerve to the diaphragm, which is uncommonly injured because of its protection from the general field of dissection by the interposition of the prevertebral fascia in the floor of the neck.

The great auricular nerve is readily located near the inferior tip of the parotid gland and the posterior border of the sternocleidomastoid muscle. Because of its size, this nerve provides an excellent donor site for nerve grafting within the head and neck.

SPECIAL CONSIDERATIONS

Patients having dissections within the floor of the mouth or tongue such as thyroglossal duct excision must be carefully watched in the postoperative period because of swelling. Swelling can proceed in a submucosal plane over the base of the tongue to occlude the larynx precipitously with possible tragic effects. Beta-hemolytic streptococcal infections following injury or surgery in the floor of the mouth can proceed rapidly with submucosal edema (Ludwig's angina) and precipitously cause laryngeal edema in the same manner. When upper respiratory obstruction presents itself in the early postoperative period, orotracheal intubation may be impossible and the surgeon must be prepared to perform a cricothyrotomy or tracheostomy.

Sialoma and Salivary Fistula Following Parotid Surgery

Occasionally, following superficial parotidectomy, a soft swelling occurs under the skin flap of the parotid dissection and upon tapping yields a fluid which is essentially parotid secretion and this can be verified by testing for parotid enzymes. Treatment comprises the periodic centesis and compression dressings, usually with resolution of the accumulation within a few weeks of its first appearance. Occasionally a fistula will arise from the site of parotid surgery implying active secreting parotid tissue. With a persistent fistula, tumor is ruled out only by reoperation and total parotidectomy. Fistula formation, however, may occur in surgery for benign parotid disease and some surgeons have chosen low dose irradiation with the accompanying radiation risks to ablate the remaining parotid tissue.

All of the aforementioned complications in head and neck surgery may be unavoidable. However, it is obvious that knowledge of anatomy, physiology, careful surgical technique, and meticulous postoperative care will minimize the number and severity of those complications.

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Thyroidectomy, Parathyroidectomy, and Modified Neck Dissection

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This article is devoted to complications which occur commonly in thyroidectomies for Graves' disease and thyroid neoplasms, in parathyroidectomies, and in modified neck dissections for metastatic thyroid cancer, with emphasis on their *prevention*. Operations designed to correct thyroid and parathyroid disorders are characterized by: (1) complex endocrine and metabolic dimensions and (2) problems related to the preservation of an adequate airway during the immediate postoperative period. General principles and problems primarily related to the anatomic location of these glands are discussed first. The management of problems related to specific endocrinopathies are discussed subsequently.

GENERAL PRINCIPLES

Preoperative and Preventive Measures

Great emphasis is placed on the following preventive measures: discontinuation of smoking 4 weeks prior to operation, atraumatic endotracheal intubation, avoidance of an esophageal stethoscope during the operation, meticulous hemostasis, and close observation during the first postoperative day.

Meticulous hemostasis is not only a sine qua non of the necessarily punctilious operative technique required in these operations, but it also minimizes the incidence of a number of related postoperative complications. For example, a 50 to 100 cc bleed in an abdominal operation may

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escape unnoticed; however, when it occurs in the neck, it may produce airway obstruction by direct pressure on the larynx or trachea and by triggering laryngeal edema.

Another important preoperative measure highly desirable in nearly all patients requiring thyroid and parathyroid operations (especially in patients with Graves' disease) is the administration of TSH suppressive doses of thyroxine for several weeks, if not months, prior to thyroidectomy. This will produce a marked reduction in the size and vascularity of the gland and consequently facilitate dissection of the recurrent nerves and parathyroids, when indicated, and minimize postoperative bleeding.

Position of Patients on the Operating Table

The head is positioned with precaution to avoid excessive hyperextension of the neck, especially in patients with cervical arthritis related to hyperparathyroidism. The head and torso are elevated to an angle of 30 degrees. The legs are also slightly elevated to avoid pooling of blood in the lower extremities. Thromboembolism stockings or Ace bandage wraps of the lower extremities are desirable. The elevation of the head and torso will provide for a drier operative field by reducing hydrostatic pressure in the venous circulation and will reduce the chances of air embolism, especially if the patient's respirations are controlled.

Endotracheal Intubation

In most institutions thyroidectomies and parathyroidectomies are performed under general anesthesia with endotracheal intubation and controlled respiration. This maintains a positive intrathoracic pressure in all phases of respiration and thus minimizes the chances of air embolism. In elderly patients and individuals with cervical arthritis (a common occurrence in hyperparathyroidism), hyperextension of the neck for intubation or operation may not be feasible, and may indeed be *contraindicated.* The anesthesiologist should be prepared for technical difficulties in intubation with a prism or a flexible laryngoscope.

If intubation is not successful after repeated attempts, the patient is awakened; the operation is rescheduled for *another day.*

In professional singers a pharyngeal airway is substituted for endotracheal intubation.

Drains

Soft rubber drains are indicated when there is a large dead space, when hemostasis is less than absolute, or when a large surface of the thyroid has been transected, as in bilateral subtotal resection for Graves' disease. The drain is placed on the surface of the remaining thyroid tissue or in the dead space, passed through an opening fashioned in the sternocleidomastoid muscle between the sternal and clavicular heads. It exits at the lateral end of the skin incision. These drains are usually removed within 24 hours. The skin edges, at the drain site, are approximated by tying a suture previously placed in the operating room and left untied. Drains are not substitutes for painstaking hemostasis and will *not* prevent the development of a hematoma. However, they will allow

for drainage of serosanguinous fluid and, if unusual amounts of bloody drainage are noted, may provide a clue to the imminent development of a hematoma and airway obstruction.

Patients undergoing parathyroidectomy for renal (secondary) hyperparathyroidism, who have been on chronic dialysis and may be prone to fluid overload, may have unusual degrees of serous drainage in the immediate postoperative period. Soft rubber or other types of drains are mandatory in these patients.

SUCTION TYPE OF DRAINAGE. Following radical neck dissection, subplatysmal space drainage is established with a plastic catheter with multiple perforations, connected to a continuous suction system. These drains exit through separate stab wounds lateral to the incision. Such catheters are removed within 24 hours. In patients with extensive node metastases such a system may not produce any significant drainage and may indeed be superfluous, since a collateral lymphatic and venous circulation may have already developed.

GENERAL PROBLEMS

Corneal Abrasions

Since the operating team and the anesthesiologists are all crowded together over a small field, the patient is especially vulnerable to corneal abrasion. This is a particular concern in patients with exophthalmos. The eyelids should be taped and protected with superimposed eye pads. A corneal abrasion will be manifest in the recovery room, when the patient complains of severe pain and photophobia. An ophthalmologist should be consulted. Treatment usually consists of topical antibiotics and an eye patch. The symptoms wane within 48 hours. On follow-up examination the ophthalmologist usually finds no sequelae.

Air Embolism

Although air embolism seldom occurs, it is always a potential complication in the head and neck operations. Controlled positive pressure ventilation through a cuffed endotracheal tube minimizes this risk. Other ancillary measures that help early detection and treatment include a centrally placed venous catheter and a Doppler ultrasound stethoscope which will detect immediately changes in heart sounds before arrhythmia or cardiac arrest occurs.

If air embolism is suspected, the head of the table is lowered immediately and the patient turned on his left side to allow the air to settle in the apex of the right ventricle; at the same time air is suctioned through a CVP catheter.

If cardiac arrest occurs the chest is opened, the air aspirated directly from the right ventricle, and cardiac massage performed, along with appropriate measures of cardiopulmonary resuscitation.

Hemostasis and Postoperative Bleeding

In order to minimize operative bleeding the patient is positioned with the head and thorax elevated at a 30° angle, as described above.

This reduces the hydrostatic pressure within the veins of the neck. However, precautions must be taken not to accentuate the hyperextension of the neck, when the thorax is elevated.

To avoid excessive intra- and postoperative bleeding, the emphasis is on dissection and technique. Veins which appear minuscule under general anesthesia and with the thorax and head elevated, will distend and cause significant bleeding postoperatively if they are not ligated.

We prefer the technique of dissecting major vessels, suture-ligating each end (single or double suture-ligatures), cutting the sutures and then the vessel itself, in that exact sequence. In other words, the vessel is not transected until it is suture-ligated at both ends. In head and neck operations, this technique is considered preferable to the placement of hemostats on various vessels, as dissection proceeds, and ligating them periodically. We do *not* advocate hypotensive anesthesia to minimize bleeding and reduce operating time: many vessels which should be ligated are overlooked in such a setting, only to cause postoperative bleeding. Furthermore, when normotension is restored prior to closure of the incision to find and clamp bleeding points and ligate them, the surgeon runs an increased risk of injuring the recurrent nerves and the parathyroids.

The most frequent sources of troublesome bleeding during thyroidectomies are the short vessels that supply the posteromedial aspect of the gland, the short anastomotic channels from the thyroid which penetrate the cricothyroid membrane and surround the recurrent nerve. Hemostasis becomes a most difficult task when those short vessels are not suture-ligated (with fine sutures and swedged-on needles) prior to transection.

In subtotal thyroidectomy, especially in patients with Graves' disease, absolute hemostasis with suture-ligatures must be obtained on the transected surface of the thyroid; this may be particularly difficult in patients whose gland has not achieved a firm consistency with prolonged preparation with thyroid blockers and thyroxine.

The superior thyroid vessels usually do not present any problem provided they are dissected and their short branches to the cricothyroid muscles are divided separately. To control bleeding from the superior thyroid artery the surgeon may have to extend the incision and dissect the external carotid to expose the origin of the superior thyroid artery.

In radical neck dissections, injuries to the origin of the internal jugular vein or to the subclavian are repaired primarily. The midportion of the clavicle may be excised to obtain adequate exposure.

Postoperative bleeding is usually manifest during the first 16 hours. This may be heralded by excessive bloody drainage from the drain site, increase in size of the neck, or distention of the neck veins. Bleeding, even in small quantities, may lead to laryngeal edema and acute airway obstruction. Treatment consists of immediate decompression by release of the skin and deep (cervical) fascial sutures at the bed side. Although endotracheal intubation may be attempted at the bed side, it is a very difficult task even in the most experienced hands; valuable time may be lost in such unsuccessful attempts. It may be simpler

and faster to insert an endotracheal tube through a direct opening in the trachea. Only then the patient is transported to the operating room, the source of bleeding controlled (usually no major bleeder is found), and the tracheostomy completed. The wound is closed loosely around the tracheostomy tube, which is removed a few days later. The wound is allowed to close secondarily.

When airway obstruction is not an immediate problem, the patient with an expanding hematoma is transferred to the operating room, the wound is reopened under sterile conditions and the hematoma evacuated. Endotracheal intubation and general anesthesia are usually required to complete the exploration and control the bleeding. Again major bleeders are seldom found. A common error in judgment is to assume that control of the bleeding is sufficient, only to see the patient progress to complete airway obstruction from laryngeal edema during the ensuing few hours, if not sooner. Therefore, in any patient who requires a second formal exploration to control bleeding, tracheostomy is considered a mandatory final step.

Airway Obstruction

This is the most serious complication following head and neck operations. Usually, it occurs within the first 24 hours. Symptoms may include dyspnea, stridor, cyanosis, restlessness, agitation, disorientation, and retraction of the intercostal spaces.

The obstruction may be sudden and nearly complete; this is usually caused by aspiration, pressure on the trachea from a rapidly expanding hematoma, or posterior displacement of the tongue. On the other hand, airway impairment caused by laryngeal edema and/or vocal cord paresis may proceed more slowly and insidiously. The first symptoms may be agitation, irritability, and then intercostal retraction. Laryngeal edema may be secondary to a variety of causes, which include difficulties in endotracheal intubation, allergy, postoperative bleeding, failure to release the balloon cuff of the endotracheal tube before extubation, and a pre-existing upper respiratory tract infection.

If the patient becomes restless, agitated, or disoriented, one should assume a hypoxic state until proven otherwise. An adequate airway is immediately established by endotracheal intubation and/or tracheostomy at the bed side or preferably performed in the operating room, if time permits. Tracheal intubation may be a very difficult task in the presence of laryngeal edema. In contrast, tracheostomy is a relatively simple procedure following thyroidectomy.

Prolonged intubation should be avoided in the treatment of this kind of airway obstruction, since it may only aggravate the laryngeal edema. A tracheostomy is preferable by far.

Unilateral vocal cord paresis without laryngeal edema is seldom a cause of airway obstruction. In rare instances of bilateral recurrent laryngeal nerve injury, with intact superior laryngeal nerve function, immediate tracheostomy is mandatory. Even if there is coexistent superior laryngeal nerve injury, a tracheostomy may still be required.

Chylous Fistula

In the course of a radical neck dissection several small lymphatic vessels are transected. The resulting lymphatic drainage usually subsides within a few days. Suction catheters represent a significant advance in the treatment of such problems by eliminating dead space fluid accumulation and reducing the incidence of wound infections.

Failure to recognize injury to large lymphatic channels may result in a troublesome chylous fistula. Since the thoracic duct drains both lower extremities, the abdominal wall, most of the abdominal viscera, and the left half of the thorax, if injured, it is more likely to cause a fistula than the right lymphatic trunk, which drains a smaller field and consequently carries a lesser volume of lymph. Identification and ligation of these trunks prior to transection avoids fistula formation.

A lymphatic fistula may produce fluid losses averaging 1500 to 2000 ml per 24 hours. This fluid contains electrolytes, glucose, and proteins in roughly similar concentrations to that of plasma. The lipid content is higher, however.

If the fistula does not show evidence of decreased output with supportive measures (i.e., electrolyte and fluid replacement; high-protein, low-fat diet; pressure dressings) after a few weeks, exploration and identification and ligation of the injured duct should be considered. If this is not successful, intrathoracic ligation of the duct may be attempted.

Injury to Recurrent and Superior Laryngeal Nerves

The *recurrent laryngeal* nerve ascends in the tracheoesophageal groove, passes behind, in front of, or between branches of the inferior thyroid artery with variable frequency, and penetrates the cricothyroid membrane just posterior to the cricothyroid articulation. It innervates the intrinsic musculature of the larynx.[1, 11, 18] It is common for the nerve to divide before penetrating the cricothyroid membrane. Thus, the anterior branch may enter the larynx in a course anterior or posterior to the cricothyroid articulation. In less than 1 per cent of instances the nerve may arise from the vagus at the level of the superior pole of the thyroid and enter directly at the cricothyroid articulation, without "recurring."[7, 9, 20] This nonrecurrent laryngeal nerve may be mistaken for the inferior thyroid artery or included in the ligatures of the superior pole vessels.

The superior laryngeal nerve arises from the nodose ganglion of the vagus and divides into an internal branch and an external branch. The internal branch enters the thyrohyoid membrane to provide sensory innervation to the mucous membrane of the larynx and epiglottis and anastomoses with branches of the recurrent laryngeal nerve. The external branch courses caudad to provide motor innervation to the inferior constrictor m. of the pharynx and passes below the oblique attachment of the sternothyroid m. to innervate the cricothyroid m., which changes the position of the thyroid cartilage in relation to the cricoid in such a fashion as to lengthen or tense the vocal cords. Injury to the external branch of the superior laryngeal nerve produces voice changes which may go almost unnoticed and may vary from mild hoarseness to inability to reach high pitched sounds or easy fatigability in speaking in

crowds. These symptoms are more pronounced with bilateral injury. Laryngoscopy may disclose a wavy outline of the cord or obliquity of the glottic aperture, or the injured side may lie at a lower level than the normal side. Rare instances of injury to the internal branch produce an inability to perceive the entrance of foreign body into the larynx and symptoms of aspiration upon deglutition; aspiration pneumonia may result.[5, 14] A high or blind ligation of the superior pole of the thyroid is likely to injure the superior laryngeal nerve, particularly when one considers that in 20 to 30 per cent of patients it passes through the capsule of the thyroid or is intimately related to the superior thyroid vessels. The frequency of injury to this nerve may be reduced by transection of the sternothyroid muscle close to its insertion on the thyroid cartilage; this usually provides good exposure for individual ligation of the superior pole vessels.

Injury to the recurrent laryngeal nerve (RLN) brings the vocal cord on the affected side to the paramedian position due to the adductor effect of the cricothyroid m. If the superior laryngeal nerve (SLN) is also injured this action is lost and therefore the cord remains in an intermediate or "open" position. The clinical presentations can be classified according to particular combinations of injuries:

a. *Unilateral recurrent laryngeal nerve injury*—the cord remains fixed in the paramedian position. The combination of the tensor effect of the cricothyroid m., the compensatory movement across the midline of the uninvolved cord, and fibrotic changes in the paralyzed cord, all tend to approximate the two cords and thus produce a good voice. Return to a functionally normal voice can be dramatically accelerated by injection of Teflon into the paralyzed cord.[4, 12]
b. *Unilateral injury to the RLN and SLN*—the cord is in intermediate position: hoarseness and inability to produce an effective cough are the principal symptoms. In time fibrotic changes will move the cord towards the midline; this and the compensatory movement of the normal contralateral cord, across the midline to meet the paralyzed one, will combine to produce an almost normal voice.
c. *Bilateral RLN injury with intact SLN*—the adductor effect of the cricothyroid muscles fixes both cords in a paramedian position, and produces near complete airway obstruction. An immediate tracheostomy is mandatory.
d. *Bilateral RLN and SLN injury*—the cords will be paralyzed in the intermediate position. The airway may be adequate, and the patient will have a hoarse, breathy voice, which improves with the development of fibrosis. The improvement of the quality of the voice in such instances may be an ominous sign, since it signifies that the cords are shifting toward the paramedian position and heralds an impending airway obstruction: again a tracheostomy may be necessary at a moment's notice.[11, 18] In bilateral nerve injury tracheostomy is usually required immediately. If there is no return of cord function in 6 to 12 months, the classic measure of laryngofissure and laterofixation of one vocal cord may be required to maintain an adequate airway; the tracheostomy tube is then removed, and Teflon or glycerin injection may further improve the dysphonia and airway problems.[4, 12] Recently, considerable success with a nerve-muscle pedicle implant in several cases has been reported by Tucker.[22] One of the strap muscles, with its motor nerve from the ansa hypoglossi, is transposed and implanted into the posterior cricoarytenoid muscle. This technique restores an adequate airway within a few weeks and thus obviates the need for a permanent tracheostomy or arytenoidectomy.

In experienced hands, injuries to the RLN are infrequent, provided it is exposed and partially dissected with great respect for its propensity to injury: it should be minimally handled, not subjected to tension and suc-

tion, and protected from drying. If injury to the main trunk or one of its two branches is noted in the course of dissection, the cut ends are approximated with 6-0 or 7-0 sutures. In half of these patients good function returns within 12 to 16 months.[15, 17] In the postoperative period the clinical presentation of a damaged nerve may be varied, as mentioned previously. The first clue is inability of the patient to produce an effective cough in the recovery room; subsequently, inability to project the voice or to phonate high pitched sounds will be noted. Sometimes RLN palsy occurs late in the postoperative course: this may be due to edema or compression of the nerve; function returns in almost all such instances within a few months.

Hypocalcemia

The incidence of hypocalcemia following thyroidectomy in general varies between 2 and 13 per cent.[2, 16, 21, 23] After total thyroidectomy it may be as high as 33 per cent,[8, 23] if attempts to preserve the parathyroids by dissection or autotransplantation are not made.[16]

There are a number of reasons for the development of hypocalcemia, as suggested by several authors. Following total thyroidectomy it is due to either the excision of the parathyroid glands or, more commonly, interference with their blood supply.

Following subtotal thyroidectomy for Graves' disease, the hypocalcemia, which is usually transient, is thought to be due to preoperative suppression of parathyroid activity by the hypercalcemia of hyperthyroidism and to the rapid accretion of calcium into bone,[2, 15, 19] as soon as the gland has been resected. Bone changes occur commonly in hyperthyroidism (increased bone calcium turnover, osteoporosis),[3, 6, 10] and are responsible for the rapid accretion of calcium into these "hungry" bones following thyroidectomy and may result in symptomatic hypocalcemia within 24 hours. Hypophosphatemia, in such patients, clinches the clinical impression that the hypocalcemia is secondary to the accretion of calcium into bones. In contrast, hypocalcemia due to parathyroid malfunction is usually manifest 2 or 3 days after operation and is characterized by hyperphosphatemia.

The incidence of permanent hypoparathyroidism following total thyroidectomy has been reduced to less than 2 per cent or less by refinements in techniques[21] and employment of parathyroid autotransplantation.[16]

The symptoms of hypocalcemia range from weakness, easy fatigability, headache, nervousness, numbness, circumoral and fingertip paresthesias, muscular cramps, carpopedal spasm, and positive Chvostek and Trousseau signs, to laryngeal stridor and convulsions.

Since individual tolerance to hypocalcemia varies considerably, the patient rather than the laboratory should determine the treatment. For example, following thyroidectomy a patient with a calcium of 8.5 and phosphorus of 4.3 may be very symptomatic, especially if he/she hyperventilates frequently or has coexisting hypomagnesemia and therefore requires aggressive therapy, whereas a patient with a calcium of 7.8 and phosphorus of 4.8 may be perfectly asymptomatic and remain so until these values stabilize spontaneously.

TREATMENT. The hypocalcemia that follows subtotal thyroidectomy for Graves' disease responds well to dietary calcium supplements in the form of milk or calcium carbonate tablets, administered every 6 hours; occasionally calciferol may be required temporarily to enhance calcium absorption. Exogenous phosphate may be temporarily restricted by dietary means or by administration of aluminum hydroxide gel, which binds inorganic phosphate in the gastrointestinal tract.

In rare instances of prolonged and symptomatic hypocalcemia or repeated episodes of mild tetany, hospitalization is shortened if the patient is treated with dihydrotachysterol (AT-10) and calciferol (vitamin D). These two sterols are started simultaneously at respective doses of 0.375 mg every 6 hours and 100,000 units daily. Calcium carbonate tablets are added to assure availability of calcium. Dihydrotachysterol provides immediate symptomatic relief with return of serum calcium to the normal range within 24 hours. The full effect of calciferol, which mainly enhances calcium absorption from the digestive tract, is not fully manifest for 10 to 14 days, at which time the dihydrotachysterol is discontinued. At this point the dosage of calciferol is reduced to 50,000 units a day. After a few weeks of normocalcemia on this regimen, the administration of calciferol can be discontinued by progressive reduction of the dose over a period of several weeks.

Following total thyroidectomy, hypocalcemia and hyperphosphatemia are the result of hypoparathyroidism, which may be temporary or permanent. If at operation the parathyroid glands were autotransplanted,[15, 16] the hypoparathyroidism is usually temporary. The implants will develop a new blood supply and parathyroid function will usually return within 3 months. Very rare episodes of full blown tetany in the immediate postoperative period are usually precipitated by hyperventilation: these are treated with a slow intravenous infusion of 10 per cent calcium gluconate; two or three 10 ml ampules, administered over a period of 15 minutes, may be required. Such a solution is administered through a large vein. In refractory cases, or in situations where dihydrotachysterol cannot be administered orally, a continuous infusion of 1 per cent calcium gluconate may be required.[8, 13]

In patients receiving digitalis, intravenous calcium should be given with great caution and *very slowly*, preferably with an electrocardiographic monitor, since calcium augments the effect of digitalis and may provoke a digitalis toxic arrhythmia. Finally, commercially available concentrations of calcium chloride should not be used, since this salt produces marked inflammatory changes at the site of administration. It may be infused in *low* concentrations via a large vein.

Thyroid Storm

Thyroid storm or crisis is a rare complication of thyrotoxicosis. The few instances occur in patients with untreated thyrotoxicosis who are subjected to stress, infections, diabetic ketoacidosis, trauma, and emergency surgical procedures. Since patients operated for Graves' disease in this day and age are well prepared, it is seldom seen in that setting.

The early signs of thyroid storm are increased toxic reaction with fever, tachycardia, and increased central nervous system activity. It may

progress to somnolence, stupor, and coma. Frequently these symptoms are accompanied by congestive heart failure, pulmonary edema, jaundice, and diarrhea, with severe abdominal pain, nausea, and vomiting which complicate the clinical presentation and suggest an acute abdominal emergency.

The pathophysiology of thyroid storm is not well understood. Circulating thyroid hormones increase in the situations described above. This results in an excessive generation of heat, which can no longer be dissipated by the usual mechanisms of peripheral vasodilatation and diaphoresis. The temperature rises rapidly. Excessive lipolysis has been postulated as the cause for the overproduction of heat in thyroid storm. Indeed the lipolytic effects of catecholamines may be enhanced by thyroid hormones. However, the interrelationship between thyroid hormones and catecholamines is not clearly understood. Although overall catecholamine metabolism seems unaltered in thyrotoxicosis and the exact role of catecholamines in thyrotoxicosis awaits further study, the belief that catecholamines contribute to symptoms of thyroid storm has led to the successful use of drugs that deplete or block catecholamines, as adjunctive therapy for hyperthyroidism and thyroid storm.

The diagnosis of thyroid storm may be difficult to establish. Thyrotoxic crisis is usually of abrupt onset and almost invariably occurs in patients who have been thyrotoxic before the onset of the storm and have not been adequately treated. The cardinal features of the diagnosis are fever, which may rise over 41° C, a silky smooth moist skin, marked weakness and muscular wasting, greatly accelerated heart rate markedly out of proportion to the rise in the body temperature, and cardiac manifestations such as congestive heart failure and pulmonary edema. The blood pressure is usually well maintained initially; however, shock may supervene ultimately. Central nervous symptomatology includes tremor, pronounced restlessness, emotional instability, confusion, and frank psychosis. As stated earlier, this may progress to apathy, stupor, coma, and ultimately death.

Once a thyroid storm is suspected, blood is drawn for future determinations of circulating thyroid hormone levels: treatment is started immediately, even if the diagnosis is not absolutely clear or confirmed. Usually four objectives govern therapy: (1) diagnosis and treatment of the underlying triggering condition; (2) general supportive measures; (3) reduction of the secretion and production of thyroid hormones; (4) blockage of the peripheral metabolic effects of thyroid hormones and catecholamines.

The supportive measures include replacement of fluids, glucose, and electrolytes. The hyperthermia is treated with ice packs or alcohol sponge baths and hypothermic blankets. Aspirin, which may increase the metabolic rate further in these patients, is avoided. Diuretics, digitalization, and oxygen by mask may be used for the treatment of congestive failure.

Antithyroid drugs are administered orally in high doses, such as 500 to 1000 mg of propylthiouracil or 50 to 100 mg of methimazole. After such a loading dose, the patients are maintained on 250 to 500 mg of

propylthiouracil or 25 to 50 mg of methimazole each day in three divided doses. In addition to its thyroid blocking effect, propylthiouracil inhibits the peripheral action of thyroid hormones and the conversion of thyroxine to tri-iodothyronine.

Although guanethidine and reserpine were popular at one time for adrenergic blocking, these have been replaced with beta-sympathetic blockers, such as propranolol which has gained widespread use for the treatment of thyrotoxicosis itself in addition to thyroid storm. In some instances, combined alpha- and beta-sympathetic blockade (phenoxybenzamine hydrochloride and propranolol) have been also used with claims of greater effectiveness. Finally, when all these measures do not seem to control the crisis, hydrocortisone in doses of 500 to 1000 mg a day is administered parenterally. There are several reports of increased survival of patients in thyroid crisis directly attributed to the administration of glucocorticoids, although documented adrenal insufficiency among such patients is rare.

It bears emphasis that thyroid storm is a *clinical* diagnosis. Its manifestations are quite variable. It may present as an illness of one or several organ systems, and mimic other conditions unrelated to thyrotoxic state. In spite of a number of current innovations in therapy, thyroid storm still carries a very high mortality. Enthusiasm for new modes of therapy seems to wane with time. For instance, there are two recent reports of thyroid storm developing during labor in patients maintained on propranolol!

Hypertrophic and Unsightly Scars

This unsightly complication is more apt to occur in young female patients operated for Graves' disease and especially in the black population. Keloids can be treated and reduced to flat, broad scars with applications or local injections of triamcinolone.

The disfiguring end results of the "H" incision, commonly used for radical neck dissections, is avoided by utilizing the standard low cervical "thyroidectomy" incision with an extension into the supraclavicular space and a curving line ascending in the posterior cervical region. Such an incision provides adequate exposure for modified neck dissections and an excellent cosmetic result.

SPECIFIC PROBLEMS

Hyperparathyroidism

CALCIUM SALT DEPOSITION IN KIDNEYS. Hydration and assurance of a good urinary flow must be maintained before, during, and after surgery (no catheters please), especially during the first 24 hours following parathyroidectomy. Adequate hydration will minimize the precipitation of calcium salts in the renal parenchyma and tubules. Such calcium salt precipitation is enhanced in the immediate postoperative period by both the reduced flow of urine (antidiuretic hormone effect) and by the sudden decrease in tubular reabsorption of calcium when circulating

parathormone plummets: this results in transient hypercalciuria, even though serum calcium levels may fall to subnormal values.

ACUTE PANCREATITIS. In some series a high incidence of postoperative pancreatitis has been reported. Furthermore, there are many reports of an association between hyperparathyroidism and chronic relapsing pancreatitis. For this reason opiates and meperidine for the relief of pain or for preoperative medication are to be avoided, especially since these patients seldom complain of severe pain. When pancreatitis is diagnosed, it should be treated according to conventional methods, with particular attention to circulating calcium levels and the administration of intravenous calcium when necessary.

DEPRESSIVE REACTION. The frequency of postoperative depressive reactions is decreasing, as compared to previous reports and our own experience some years ago. As a rule they are of short duration. The services of a psychiatrist are rarely required; the usual supportive measures and the surgeon's attentiveness should be quite sufficient. A continued state of borderline tetany may aggravate the patient's symptoms. Such reactions may be precipitated by rapid steroid withdrawal in patients who have received large suppressive doses of glucocorticoids during the course of their preoperative evaluation, especially when their administration has been maintained during the operative and postoperative period.

PSEUDOGOUT AND GOUT. In hyperparathyroidism uric acid levels may be elevated without any overt symptoms. Following parathyroidectomy these values usually fall into the normal range. The relationship between pseudogout and hyperparathyroidism is not clear, except for reports of a high incidence of hyperparathyroidism in pseudogout. Parathyroidectomy seems to have no influence on the course of pseudogout; in fact, acute attacks may occur in the immediate postoperative period. These are treated according to established principles.

In contrast, patients with gout and hyperparathyroidism seem to be benefited by parathyroidectomy; the acute attacks may be less frequent, and long-term treatment, other than dietary measures, may no longer be required.

Graves' Disease

Postoperative bleeding, airway obstruction, recurrent nerve palsy, thyroid storm, and severe hypocalcemia were the classic complications of thyroidectomy for Graves' disease. Today, with adequate preparation of patients over a prolonged period of time, which allows for metabolic restoration and especially the replenishment of bone calcium reserves, thyroid storm and hypocalcemia are seen rarely. Bleeding and airway problems have been discussed in the preceding pages.

EXOPHTHALMOS. There is the general, albeit unsubstantiated, observation among ophthalmologists that ocular ophthalmopathy may be precipitated by a state of hypothyroidism in patients with Graves' disease. This is induced by the administration of thyroid-blocking agents, radioactive iodine, or subtotal thyroidectomy. For these reasons, it is important to maintain these patients on therapeutic regimens that will not

allow the inadvertent development of hypothyroidism: for instance, it is preferable not to attempt to titrate these patients with graduated doses of thyroid blockers. It is best to give them complete blocking doses and to add thyroxine to the regimen. In clinics where patients are treated with radioactive iodine in large numbers, an overdose of radioactive iodine is prescribed along with thyroxine, which the patients will have to take permanently. Following bilateral subtotal resection for Graves' disease, the situation is somewhat different, since the short-term incidence of hypothyroidism is less than 5 per cent, whereas the long-term 20-year incidence may be as high as 40 per cent.

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Colostomy and Colostomy Closure

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A retrospective study was made of 181 consecutive patients subjected to colostomy from 1968 to 1976 at Northwestern Memorial Hospital. There were 79 males and 102 females; their ages ranged from 20 to 90 years with a mean age of 65 years. One hundred of the patients later had colostomy closure, 39 of them male and 61 female. Eighty-one patients had no colostomy closure; 40 of these patients were male and 41 were female. The mean age of the patients who had their colostomies closed was 61 years; and those that did not averaged 71 years. The group of patients that did not have their colostomies closed was composed mainly of patients with abdominoperineal bowel resection and those with a palliative colostomy for disseminated carcinoma. There were no infants or children as these patients are treated in an affiliated children's hospital. The number of patients with penetrating injuries is small; these patients are usually seen by a nearby municipal hospital. The operations were performed by the attending physicians or by surgical residents under the direct supervision of the attending staff. Eighty per cent of the patients were private patients and 20 per cent were service or clinic patients. Twenty-seven of the 181 patients, or 15 per cent, were operated upon as an emergency procedure—14 for obstruction from carcinoma and six for obstruction from diverticulitis; the remaining seven had a variety of conditions.

The indications for colostomy are summarized in Table 1. A majority of the patients, 100 out of 181, or 55 per cent, were operated upon for complications of carcinoma of the colon. Colostomies were performed as part of abdominoperineal bowel resection, for acute obstruction, to protect low anterior colon anastomoses, and for colovesical, colovaginal, and colocutaneous fistulas. The 15 patients operated upon for carcinoma in organs other than the colon usually had colostomy performed as a palliative procedure late in the disease or had fistulas from a combination of tumor and irradiation. Diverticulitis with its complications was the reason for colostomy in 54, or 30 per cent, of the patients. In this group, perforation with abscess was the most common indication for colostomy followed by obstruction and colovaginal fistula. Among other conditions

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Table 1. *Indication for Colostomy in 181 Patients*

Carcinoma of colon	100
Carcinoma of uterus or ovary	9
Carcinoma of urogenital tract	3
Carcinoma of retroperitoneum or mesentery	3
Diverticulitis	54
Colovaginal fistula following hysterectomy	2
Gunshot wound	2
Miscellaneous	8

that led to colostomy was a patient with rectal perforation during a barium enema, a colocutaneous fistula following a hernia repair, and several complications from granulomatous bowel disease. Only two patients had a colostomy performed for gunshot wounds.

Colostomy Techniques of Current Series

Of the 81 patients that did *not* have their colostomies closed, 39 had transverse colon colostomies, five were in the descending colon, and 32 were in the sigmoid colon. Cecostomy, known for its complications, was not used. Two cases were listed as Mikulicz colostomies and were presumably in the sigmoid colon. The site of the remaining three could not be determined from the hospital records. With the exception of single-barreled end colostomies performed in abdominoperineal bowel resection, the colostomies were usually variations of loop colostomies over an ostomy or skin bridge. Of the 100 patients who had colostomies that were later closed (temporary colostomies), 89 were described as transverse colostomies, one was a Mikulicz type and the remaining 10 were not clearly described as to type or location. While these operations were performed by several surgeons, a rather uniform method of right transverse loop colostomy over an ostomy bridge emerged as the favorite procedure. Descending colon and sigmoid colostomies were usually performed at the proximal sigmoid colon and were brought through left-sided abdominal incisions. In most instances, colostomies were opened 24 to 48 hours postoperatively. In about 10 per cent of the cases the colostomies were opened at the time of colostomy, the later being a recent group in which the peritoneum and fascia were closed tightly around the colostomy, the small opening preventing peritoneal soilage when the colostomy was opened.

Patients with temporary colostomy were usually fitted with an appliance to collect the discharge. Patients with permanent colostomies were usually instructed in methods of irrigation by a stomal nurse; the only patients in this group who continued to use external appliances were patients ill with terminal disease or who were too infirm to learn irrigation techniques.

Colostomy Complications of Current Series

There was one death following colostomy—a mortality rate of 0.6 per cent. This was a 62-year-old woman operated upon for carcinoma of

the rectum. This death was 5 weeks following colostomy and was due to peristomal evisceration, cardiac arrest, and later sepsis. There were 50 other patients with significant problems following colostomy, giving a complication rate of 28 per cent (Table 2). The complication rate was 37 per cent in those patients with permanent colostomy and 21 per cent in those with temporary colostomy. The most frequent complications were related to making the opening either too tight (12 cases) or too loose (24 cases). A stoma that was too tight led to edema of the bowel (one case) and late stenosis (11 cases). An opening that was too loose resulted in evisceration (one case), prolapse (ten cases), redundant mucosa (five cases), peristomal hernia (six cases), obstruction of the small bowel (one case), and retraction of the colostomy (one case). Wound infection was seen in three cases; recurrent stomal tumor occurred in three cases and sepsis caused death in one case. Other complications such as stress ulcer, embolus, and myocardial infarction accompany all operations and are proportional to the degree and severity of the operation and to the pre-existing illnesses. The psychological problems could have been reduced by improved preoperative counseling. It is quite apparent that that *most* serious complications result from too wide a peristomal opening. The late complications of colostomy stenosis, while an inconvenience, were easily handled by minor revision.

Colostomy Closure Techniques of Current Series

The median time between performance of colostomy and colostomy closure was 24 weeks in patients with carcinoma and 47 weeks in patients with diverticulitis. These times were increased by physician or patient delay in a few cases—one patient went 3 years and another 4 years before closure. A majority of the patients had a partial colectomy performed during this interval. Prior to closure of the colostomies the patients usually had sigmoidoscopic and barium enema examinations. A

Table 2. *Complications After Colostomy in 181 Patients*

Stenosis requiring revision or dilatation	11
Prolapse requiring revision	10
Peristomal hernia	6
Redundant mucosa requiring revision	5
Wound infection	3
Recurrent tumor at stoma	3
Severe psychological problems	3
Skin excoriation (severe)	2
Evisceration	1
Small bowel obstruction	1
Retraction of colostomy	1
Severe edema of loop	1
Myocardial infarction	1
Pulmonary embolus	1
Stress ulcer	1
Sepsis (death)	1
TOTAL	51 (28 per cent)

Table 3. *Colostomies Closed*

Colon carcinoma	45 of 100
Diverticulitis	42 of 54
Gynecologic carcinoma	3 of 9
Urologic carcinoma	1 of 3
Retroperitoneal carcinoma	0 of 3
Gunshot wound	2 of 2
Following hysterectomy	2 of 2
Trauma (rectal tube and hernia)	2 of 2
Granulomatous colitis	2 of 4
Quadraplegic with obstruction	0 of 1
Cholecystocolic fistula	1 of 1
	100 of 181

bowel preparation was carried out in a variety of ways, but it usually included a liquid diet for 2 days, oral neomycin, and oral tetracycline or erthromycin. Those patients whose colostomies were closed are listed in Table 3. It is noteworthy that less than half of the colostomies in patients with carcinoma have been closed. It is presumed that a few of these patients may be closed at a later date; only 6 months has elapsed since the study was completed. The patients who had their colostomies taken down as part of a second stage colon resection are not included in our closure group. The technique of colostomy closure was by open, two-layer closure in the majority of the cases, a closed, one-layer closure being used in only a few patients. In about 15 per cent of the cases, the loop colostomy was divided completely, or the exteriorized loop was resected prior to end-to-end anastomosis. The latter was often seen in patients with prolapsed or necrotic colostomies, and these patients had a somewhat higher complication rate. In most cases, the colon was completely freed of attachments and dropped below the peritoneum. The abdominal wall was usually closed with nonabsorbable sutures, and the skin and subcutaneous tissues were usually left open. Most patients did not receive postoperative antibiotics, the exceptions being those with serious systemic diseases.

Colostomy Closure Complications of Current Series

There was one death, a 70-year-old woman, who had been operated upon for diverticulitis. She developed a colocutaneous fistula and pneumonia; she had a cerebrovascular accident and died of sepsis 2 months after the colostomy closure. Two patients had small bowel obstructions; one was relieved by reoperation and one by intubation. Two patients had large bowel obstruction; one was relieved by recolostomy and one by a prolonged liquid diet. Two patients had wound infections that required drainage, both having had primary cutaneous closure. There were probably other patients with minor subcutaneous wound infections, especially in patients with delayed closure, but this could not be discerned from the hospital or office records. Seven patients developed incisional hernias, most of them occurred within a year of colostomy closure. Two

of these patients extruded the nonabsorbable sutures prior to the herniation. One other patient developed a fecal fistula, but this closed spontaneously in about a month. Diarrhea following colostomy closure has been described and it occurred in one of our patients.[21] The complications of colostomy closure are listed in Table 4. We had a 17 per cent complication rate and a 1.0 per cent mortality rate.

DISCUSSION

Ever since the first successful colostomy (actually a cecostomy) was reported by M. Pellore in 1776, the complication rate has been significant. The morbidity rate for colostomy is usually reported as around 20 to 30 per cent.[4, 6, 18] The mortality rate directly related to the colostomy is about 1 per cent.[5, 8, 19] The unrelated hospital death rate is much higher, as many patients have a colostomy performed during a terminal illness. The complication rate is highest in cases that are done under emergency conditions, in sigmoid colostomies, in cecostomies, in end colostomies, or when the colostomy is placed in the incision. Most complications are noted within 3 years. The reported complication rates for colostomy closure have ranged from 10 to 44 per cent.[2, 3, 9, 14, 20, 23, 24] The mortality rate has been reported to be from 0 to 3 per cent.[14, 16, 20, 23, 24] In our series the complication rate of colostomy was 28 per cent and the mortality rate was 0.6 per cent. In our patients who had colostomy closure the complication rate was 17 per cent and the mortality rate was 1 per cent. When the risks of both procedures are added the complication risk is not unlike the results reported in primary colectomy.[7] Of our 100 patients who had both procedures, 35 had complications, three of whom had problems following each operation. The complication rate in our series is similar to other studies reported in the literature and would appear to be about the same whether the colostomy is performed for injury or for medical illnesses. These complications could be reduced if colostomy is performed only when absolutely indicated or if development of better techniques for colostomy and colostomy closure occurs. There have been reports in the surgical literature suggesting that the indications for colostomy should be reevaluated.[1, 15, 24] There are only two basic reasons

Table 4. *Complications of Colostomy Closure in 100 Patients*

Incisional hernia	7
Large bowel obstruction	2
Small bowel obstruction	2
Wound infection	2
Fecal fistula (nonfatal)	1
Diarrhea	1
Thrombophlebitis	1
CVA, fecal fistula, pneumonia, sepsis (death)	1
	17 (17 per cent)

for colostomy. The first is for permanent colostomy to replace the anus. This procedure is usually used in malignant disease; the indications are rigid and alternate procedure unlikely. The second reason for colostomy is to divert temporarily the fecal stream for an obstructed or damaged colon. Primary repair of small colonic injuries without protective colostomy has been suggested;[10, 15, 17] it has been suggested that fewer colostomies are needed to protect low colon anastomoses.[1] There has been a trend toward primary resection of obstructed colon lesions as well as of perforated colonic lesions, both without colostomy.[1, 10, 15, 17] Since it is unlikely that the indications for colostomy will be significantly reduced in the near future, it would appear that the complication rate could best be lowered by improved planning of the colostomy, better techniques of performance of the colostomy, and more thoughtful postoperative care.

The complications of colostomy would appear to be related to the site of colostomy, the type of colostomy performed, the size of the opening in the abdominal wall, and control of infection. The site must be selected so the colon can be exteriorized without tension, with an adequate blood supply, and in a position that allows patient inspection and manipulation of colostomy appliances. From the practical standpoint this means that the colostomy should be placed in the right upper quadrant or the left lower quadrant of the abdomen. A simple loop colostomy meets almost all of the requirements for a colostomy. The Mikulicz, Rankin, Hartman, or Devine procedures have a very limited, if any, indication in current surgical practice. Complete diversion can be achieved in a loop colostomy by suturing the mucosa of the afferent loop to the skin[22] or by a pursestring suture around the efferent loop[12] (see Fig. 7). The distal opening should not be closed if venting of the distal colon is needed because of obstruction. Loop colostomy of the left colon is best performed at the junction of the descending and sigmoid colon so that the proximal loop is fixed by the peritoneal reflection thus reducing prolapse. Cecostomy and sigmoid colostomies should be avoided.

The vast majority of the complications of colostomy are related to the size of the aperture that is made in the abdominal wall. When this opening is too small, it can lead to obstruction, edema, ischemia, necrosis, or stenosis. These problems can be easily avoided by digital palpation of the pericolonic aperture at the time of colostomy and again by examining the intraluminal aperture when the colostomy is opened. Simple skin stenosis in end colostomies can often be avoided by a circular rather than a linear colostomy skin incision. Late skin or colostomy stenosis can be corrected under local anesthesia as an office procedure. Stool impactions in small openings can be avoided by controlling the dietary liquids and by using stool softeners. The problems created by too large a peristomal defect are more common and are more difficult to treat. Such large openings can lead to evisceration, peristomal small bowel herniations with obstruction, peristomal hernias, prolapse of redundant mucosa, retraction with incomplete diversion of the fecal stream, and prolapse (Fig. 1). If the ostomy bridge is removed too early in patients with a loose opening, the colostomy may retract completely into the abdominal cavity requiring emergency operation.

Figure 1. Prolapse of transverse loop colostomy with early necrosis.

Retraction of an end colostomy can be avoided by clamping each side of the exteriorized colon with Kocher clamps at the time the obstruction clamp is removed; these clamps should remain in place 7 days. Prolapse should be manually reduced and patients should be taught this maneuver. Many of the conditions described require emergency or elective reoperation even in patients with temporary colostomy; they can be avoided by proper closure of the tissues around the colon.

Infection control is based on good mechanical and antibiotic bowel preparation. Only 15 per cent of the patients in our series were emergencies, the remainder usually had ample opportunity for colon preparation. Emergency cases, patients with severe systemic diseases, or patients with vascular prostheses should have preoperative and postoperative systemic antibiotics. Wound care should be as meticulous as in any other abdominal surgery despite the unpleasantness of fecal soilage; this care should not be relegated to nurses or ostomy therapists.

Bleeding problems can be minimized by exacting operative hemostasis and with careful passage of the Penrose drain or ostomy bridge through an avascular portion of the omentum, mesocolon, and gastrocolic ligament. Hematomas of the mesentary and bowel wall should be controlled by digital compression and evacuation of the hematoma.

Peristomal fistula formation is often related to suturing the full thickness of the bowel to the fascia or to the skin or to granulomatous bowel disease. If the fistula does not heal after a period of conservative therapy, a formal colostomy revision may be required. Colon perforation for ostomy irrigation is not uncommon if the proximal loop is redundant and not properly fixed, or if the patient has peristomal hernia. Anyone irrigating a colostomy should learn to perform a careful digital examination of the afferent loop in order to determine the course and direction of the bowel being irrigated. Once a perforation has occurred, the patient

will have the signs and symptoms of an acute abdomen following irrigation. Repair should be carried out through a formal laparotomy incision.

Bleeding from the colostomy can be from granulation tissue at the colocutaneous junction or from the inflammatory mucosa polyps. Both are easily treated by application of silver nitrate sticks or by electrocautery. Bleeding secondary to local trauma should be treated by gentle pressure, and the patient should be taught to do this. More serious bleeding can accompany stomal varices related to portal hypertension. This problem, while rare, can become serious in patients with permanent colostomies. Treatment is often complicated by the systemic disease and local ligation of bleeding points is only a temporary measure. Recurrent stomal tumor is a rare but serious complication that is readily diagnosed by visual inspection and digital palpation of the colostomy. Recurrent intraperitoneal tumor is less apparent but can often be diagnosed by deep digital palpation of one or both loops of the colostomy.

Malfunctioning of the stomal apparatus is usually caused by placement of the colostomy in a fold of skin rather than on a protuberant surface or by placing the colostomy too near a bony prominence. These problems can be avoided by placing the ring of the apparatus on the abdomen when selecting the site for the colostomy. Skin excoriation can be treated with appropriate creams and lotions and use of a properly fitting apparatus. The stomal discharge should be tested for acidity and alkalinity so the appropriate medication can be applied. Several varieties of karaya gum protective materials along with aluminum hydroxide and zinc oxide have been used. The use of the services of enterostomal therapists has been most rewarding for both physicians and patients.

Physiologic problems of flatus, odor, diarrhea, and constipation can be corrected by the use of a diet that eliminates gas forming foods and the judicious use of coffee and other irritants that cause bowel stimulation. The patient must be reassured that observing modest controls on liquid and solid intake, such as onions, and simple medications such as Metamucil and Lomotil will correct these problems. The patient must be warned about the side effects of other medication (such as antibiotics or antacids) on the activity of the colon. Bismuth subgallate tablets (100 mg q.i.d. orally) have been used to reduce colostomy odor, as has Banish, ten drops in the colostomy bag. This is available without prescription.

Psychological problems such as fear of offending others by colostomy odors, the depression that follows any disfiguring procedure, the fear of sexual dysfunction, and the fear of socioeconomic loss can be lessened by the physician's awareness of these problems and sound preoperative and postoperative counseling. A key to this problem is the establishment of open communication concerning these problems and attentive listening as the patient verbalizes his fears.

The complications arising from closure of colostomy are related to bacterial infection in the wound, leakage of the anastomotic line, bowel obstruction and, later, wound herniation. Good mechanical and antibiotic preparation of the colon with meticulous wound care will reduce the infection rate and the rate of subsequent herniation. Leakage from the colon suture line can be reduced by simple closure of a loop colos-

tomy without resection whether the colostomy follows war colon injuries, civilian colon injuries, or diseases of the colon.[3, 11, 20] Simple closure reduces operating time and is technically safer and easier.[3] After colostomy closure the skin and subcutaneous tissue should be left open in order to reduce the number of wound infections.[23] Fecal fistulas usually close if there is no obstruction distally and should have conservative management.[2, 14] The incisional hernia rate encountered in patients operated for cancer or diverticulitis is higher than in patients operated for injury—probably because of the older age group and the associated systemic diseases.

SUGGESTED TECHNIQUE FOR COLOSTOMY

Whenever possible the permission of the patient or the next of kin should be obtained prior to performing a colostomy. Psychologically, the patient should be prepared for the possibility of a colostomy even if the surgeon is uncertain of the exact operation that will be needed. In the preoperative counseling the lack of alternate methods of preserving the patient's life and health must be emphasized. The disfigurement and nuisance problems of colostomy must be mentioned but not dwelt upon. The temporary nature of the colostomy should be emphasized when this is indeed a possibility. The patient should go into the operating room with the full knowledge of the possible procedures so as to avoid the emotional shock of an unexpected procedure.

The preoperative bowel preparation that we prefer for elective colon resection includes 3 days of liquid diet, Fleets Phosphosoda orally the first 2 days, and a Fleets Phosphosoda enema the day before the operation followed by tap water or saline enemas. Erythromycin base (500 mg orally every 6 hours) is given for 3 days before the operation. Oral neomycin (1.0 gm orally every hour for 6 hours) is given the day prior to operation. This preparation is possible except in patients with bowel obstruction. As in any major operation, blood and electrolyte imbalances should be corrected prior to operation.

The colostomy site should be selected, usually in the right upper quadrant or the left lower quadrant of the abdomen by placing the incision over the most protuberant portion and away from bony prominences. The anesthetic should be a general anesthesia unless otherwise contraindicated. A transverse loop colostomy is performed by making a ten cm transverse incision in the right upper quadrant of the abdomen (Fig. 2). The anterior rectus fascia is opened transversely for 9 cm. Three 2-0 nonabsorbable sutures are placed in each end of the incised fascia. The rectus muscle is bluntly separated and retracted. An 8 cm transverse incision is made in the posterior rectus fascia and peritoneum. Again, three 2-0 nonabsorbable sutures are placed in each end of this fascial opening (Fig. 3). The transverse colon is identified (by the greater omentum and the tinea) and is brought out through the wound. A Penrose drain is carefully passed around the colon near the serosa in avascular portions of the omentum, mesocolon, and gastrocolic liga-

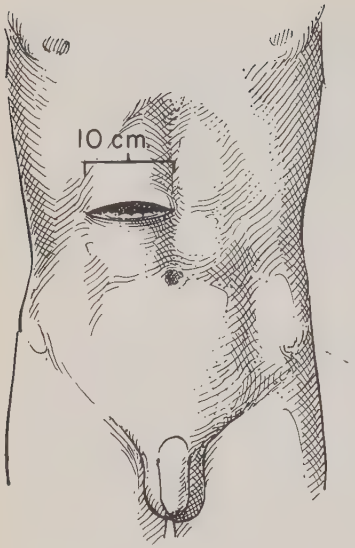

Figure 2. Recommended site of transverse loop colostomy. (Figs. 2, 3, and 5 reproduced from Hines, J. R.: A method of transverse loop colostomy. Surg. Gynec. Obstet., *141*:426, 1975. Used with permission of Surgery, Gynecology & Obstetrics.)

ments. The ends of the drain are held in a clamp and the colon dropped back into the abdomen. By pulling on the drain, the colon can be retrieved back into the wound, leaving the omentum and other structures beneath the peritoneum (Fig. 4). The posterior sheath sutures are then tied (starting with the sutures farthest from the colon) until the opening around the colon admits only the distal interphalyngeal joint of the index finger (Fig. 5). This process is repeated tying the anterior rectus sutures. The ostomy bridge (a polyethylene rod) is then placed under the colon and the Penrose drain is removed. The bowel is *not* sutured to the peritoneum, fascia or skin. The lateral margins of the skin are sutured snugly around the colon.[13] A semirigid gasket is placed over the loop of the colon on to the skin and is hooked into the ostomy bridge (Marsan

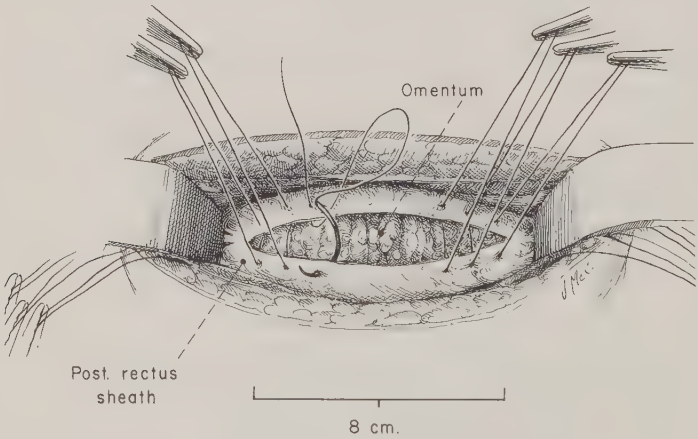

Figure 3. Sutures are placed, but not tied, in both rectus sheaths before exploring abdomen.

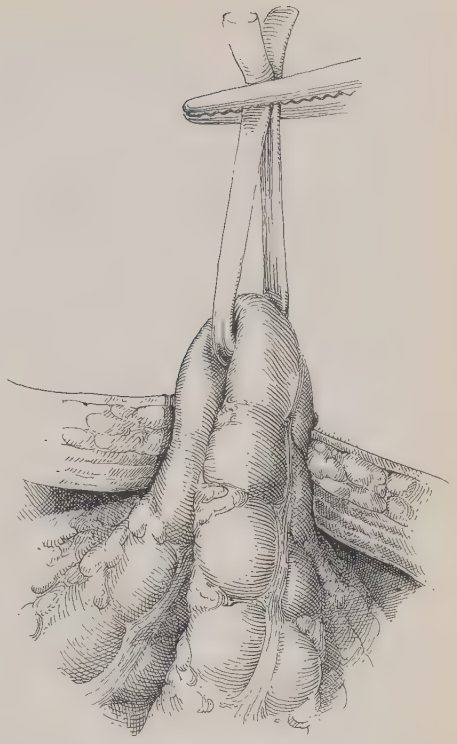

Figure 4. Following dissection, the loop of colon is retrieved by traction on the drain, leaving most of the omentum and fat below the peritoneum.

Manufacturing Co.) (Fig. 6). A karaya gum gasket should be placed under the polyethylene gasket to facilitate skin care. This colostomy is water-tight and can be opened at once if immediate decompression is indicated. A sterile collection apparatus is then applied. Vaseline gauze can be placed around the colon if it is to be opened the following day. If

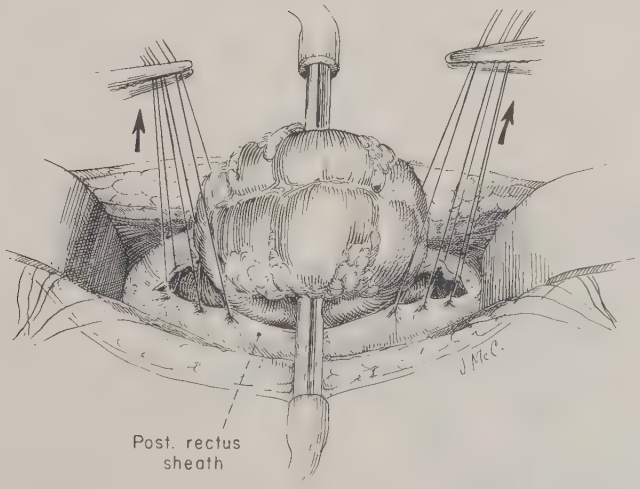

Figure 5. Sutures are tied as needed to produce snug openings in both rectus sheaths.

total diversion is indicated, a pursestring suture can be applied to the efferent opening[12] (Fig. 7). When this suture is put through all layers of the bowel wall it will effectively block stool passage for six to eight weeks. It is easily removed and does not interfere with colostomy closure.

End colostomies have three principles that must be observed: The colostomy is best performed at the start of the sigmoid colon so that the afferent loop is anchored to prevent prolapse; the blood supply must be good when the cut end is brought 4 cm above the skin; the fascia and peritoneal openings must be snug admitting only the tip of the index finger. End colostomies can be matured at the time of operation or a week later; one can wait and see if maturation is necessary.

Postoperatively, the patient has nothing by mouth for 2 days, a liquid diet for the next 2 days, and then receives a low residue diet. The ostomy bridge and skin sutures are removed on the 8th postoperative day unless poor healing dictates a longer interval. Showers are permitted 1 week after colostomy. Patients with permanent colostomies are urged to irrigate the colostomy every morning and to cover the colostomy with toilet tissue beneath an elastic waist band. Patients with temporary colostomies are trained to use the disposable collection apparatus.

The time interval between colostomy and definitive colon resection depends upon the disease present. Every attempt must be made to resect malignant tissue within 4 weeks. Resection of perforated diverticular disease requires a waiting period of 12 to 16 weeks. The time interval between the definitive colon resection and closure of the colostomy can be from 4 to 12 weeks. This allows the inflammatory reaction to subside but the distal colon opening will still be distendable. Complementary colostomies can be closed in 3 weeks; colostomies following colon injuries are closed in 4 weeks. Both groups should have appropriate study (barium enema, proctoscopy) of the distal colon prior to closure. Colostomies associated with radiation injuries or granulomatous disease may need to remain in place a year or more.

SUGGESTED TECHNIQUE FOR COLOSTOMY CLOSURE

Prior to colostomy closure, each patient should have a proctoscopic and barium enema examination. Barium introduced into the defunc-

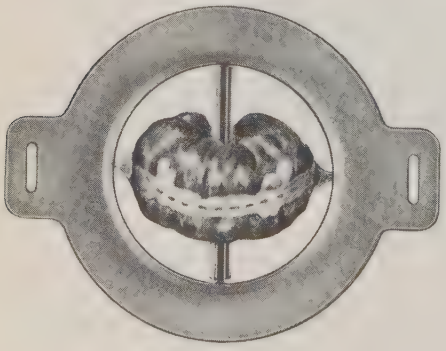

Figure 6. Rod beneath colon is attached to slots in face of gasket and is rotated to lock in place. After opening colostomy the disposable drainage sleeve is attached. (Marsan Manufacturing Co., Wausau, Wisconsin.)

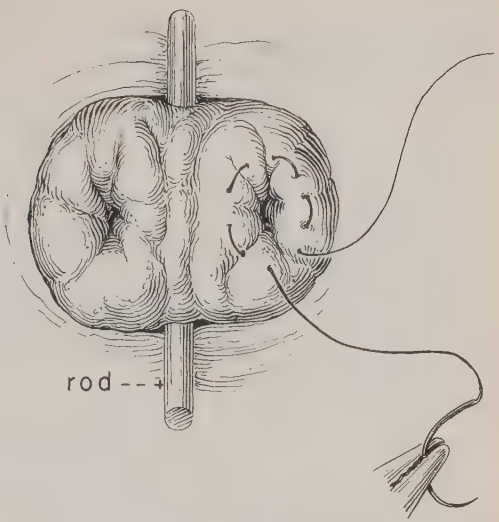

Figure 7. Method of converting loop colostomy to diverting colostomy. (Reproduced from Hines, J. R.: Techniques for conversion of a loop colostomy to a diverting colostomy. Surg. Gynec. Obstet., *140*:433, 1975. Used with permission of Surgery, Gynecology & Obstetrics.)

tionalized segment of colon has a tendency to inspissate and be retained. Preoperative bowel preparation for colostomy closure is similiar to that of elective colon resection and should include preparation of the distal loop. Thorough irrigation of the distal segment to assure complete evacuation of barium is mandatory. The small colostomy that results from the tight closure described above usually makes for easy colostomy closure without transection or resection of the loop of colon. An ellipse of skin is taken to include excoriated or granulation tissue. The ends of this skin ellipse are grasped with heavy hemostats and gently lifted in order to facilitate the dissection of the loop of colon from the fascia and peritoneal attachments. Since the bowel was not sutured, it is usually easy to dissect. The peritoneum should then be palpated with the index finger to be certain that it is free of attachments to the colon and mesocolon for the entire circumference of the defect. The skin and the cicatricial scar are then removed from the colostomy opening and the edges freshened. The colonic opening is then closed at right angles to the long axis of the colon in two layers, the inner a running 3-0 catgut Connell inverting suture, the outer interrupted 3-0 nonabsorbable Lembert sutures. At times, fibrosis, distortion, or attenuation of tissues will necessitate transection or resection with end-to-end reanastomosis. The colon is then dropped back into the abdominal cavity and the abdominal wall is closed with a single layer of heavy nonabsorbable sutures through the peritoneum and fascial layers. The skin and subcutaneous tissues are left open or loosely packed with iodoform gauze. This wound will heal by secondary intention or it can be partially closed with tape strips on the 7th postoperative day if there is no evidence of infection.

In the immediate postoperative period, the patient is given nothing by mouth for 3 days, surgical liquids for 2 days, nourishing liquids for 2 days, and is discharged on a low residue diet for 3 weeks. The postoperative care should be similar to that of any colon resection. It is a common error to treat these cases as minor surgery and this attitude has contrib-

uted to the high complication rate. The patients should be seen at weekly intervals in the office or clinic until the wound is healed.

SUMMARY

A series of 181 adult patients subjected to colostomy was studied, almost all of whom had disease rather than injury. The complication rate was 28 per cent. One hundred of these patients had a subsequent colostomy closure with a complication rate of 17 per cent. Of the patients who had both procedures, 35 (35 per cent) had one or more significant complications. The causes and prevention of these complications are described.

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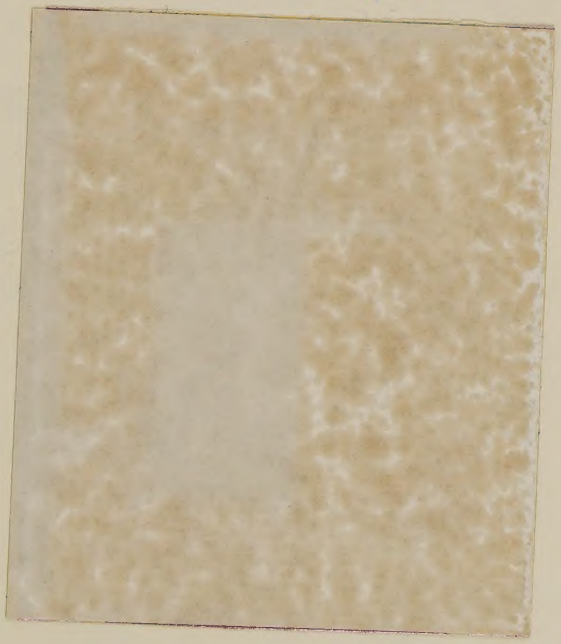

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